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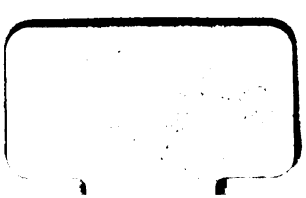
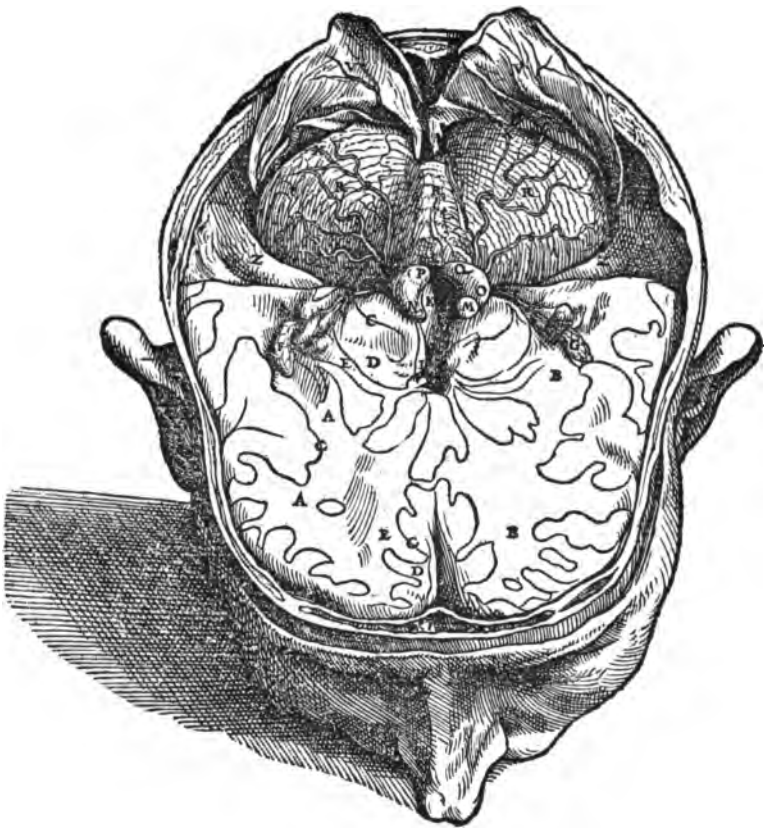


CEREBELLAR ABSCESS



THE FIRST SCIENTIFIC FIGURES OF THE CEREBELLUM, FROM
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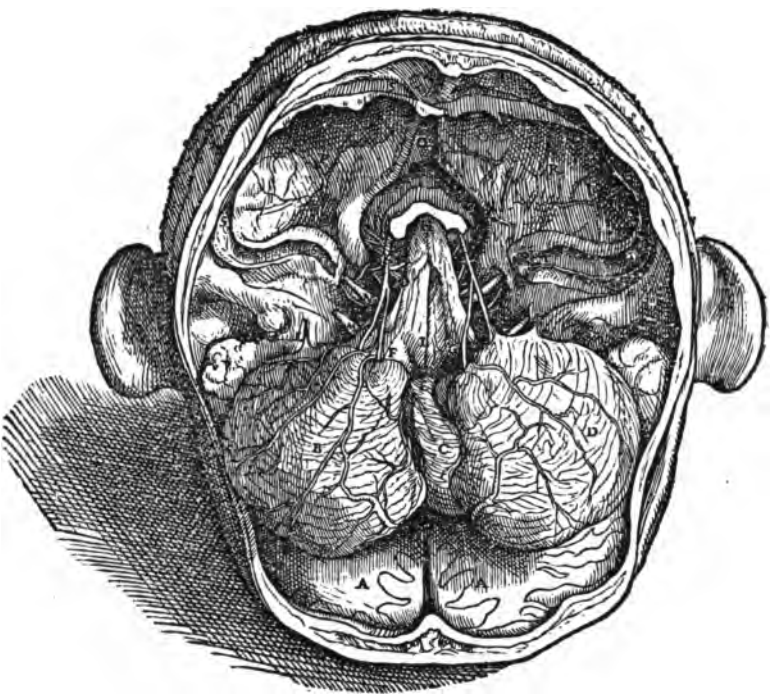

"Humani Fabrica" of Andreas Vesalius, 1555.
the Publisher by Dr. Abraham Jacobi.

CEREBELLAR ABSCESS

ITS ETIOLOGY, PATHOLOGY, DIAGNOSIS AND TREATMENT

INCLUDING

ANATOMY AND PHYSIOLOGY OF THE CEREBELLUM

BY

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WITH 10 FULL PAGE PLATES AND 16 ILLUSTRATIONS IN TEXT

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PREFACE

Ninety-eight per cent of cerebellar abscesses are otitic in origin, and the diagnosis and treatment of this complication are, to-day, almost solely the province of the otological surgeon.

Considering the recent advances made in our knowledge of cerebellar physiology and methods of diagnosis, and particularly of the relationship between the cerebellum and the static labyrinth, a work on cerebellar abscess is not untimely. This relationship we have described at length, trusting that it will interest the neurologist as well as the otologist.

In this book, we have attempted to outline briefly the anatomy and physiology of the cerebellum as a basis for that neurological knowledge with which the surgeon must be equipped in order to cope with the problem of cerebellar diagnosis. We have made no attempt to state all the opinions with regard to the physiology, but have confined ourselves to those hypotheses which seemed most plausible and which shed most light upon the subject. The etiology, pathology and symptomatology have been based chiefly upon the reports of 86 cases which we have been able to collect from the literature since 1906. The illustrations are

from original drawings and photographs made by one of us (Braun).

We have made no attempt to mention every book which has been written on this subject, but have referred only to those publications which were used in the preparation of this work.

We wish to thank Dr. T. Passmore Berens, Dr. Wendell C. Phillips, Dr. Arthur B. Duel, Dr. John B. Rae, and many of our colleagues at the Manhattan Eye, Ear and Throat Hospital, for the opportunities they have afforded us to study their cases of brain abscess.

We also wish to thank Mr. Paul B. Hoeber for many useful suggestions, and for the careful manner in which he and his staff have prepared this volume.

ISIDORE FRIESNER—ALFRED BRAUN.

NEW YORK, May, 1916.

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CHAPTER I

ANATOMY OF THE CEREBELLUM

Page 40, legend, [illegible] should be C[illegible].
Page 68, legend. Ds, Center for inward tonus of shoulder, *read* "downward" *for* "inward."
Page 77, line 12. Growers *should be* Gowers.
Page 115, legend. Pointing Test for Rotation, *read* "after" *instead of* "for."

‾IAPTER I

OF THE CEREBELLUM

m is situated in the posterior skull. It occupies most of that cranial cavity which lies below elli. The tentorium is a hori- ıe dura, attached posteriorly to the occipital bone at the level ɔphili and the horizontal portions es, and laterally to the postero- the petrous pyramids. Its ante- ntic in shape and free, and forms ıry of an opening for the passage It separates the middle from the he skull. The occipital lobes of ı its upper surface; the cerebellum s lower surface.

e of the cerebellum lies upon that ital bone below the groove for the niddle portion lies above the mid- edulla. Laterally it is in relation surface of the petrous pyramids. proximity to the internal ear. The portion of the cerebellum is in

contact with the inner table limiting the mastoid antrum and cells.

Between the layers of this portion of the dura runs

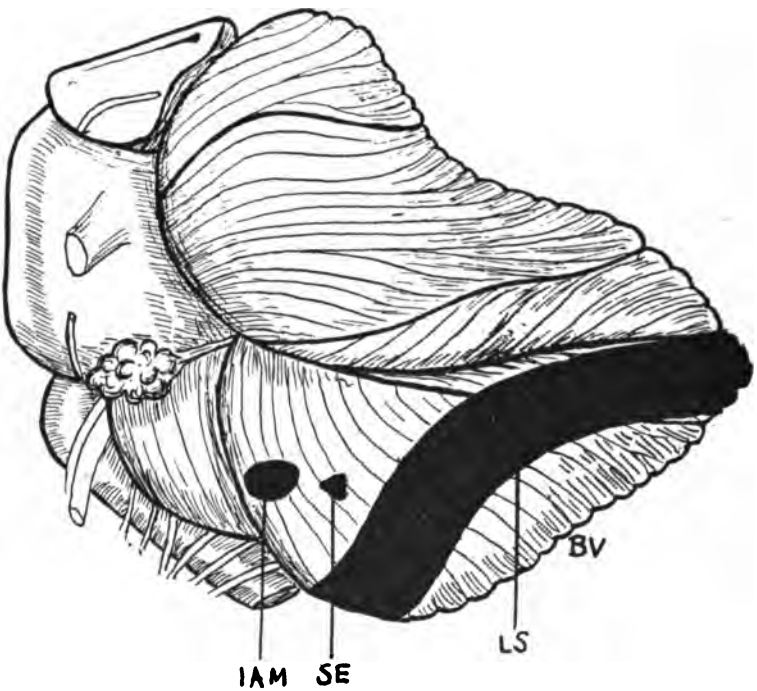


FIG. 1.—RELATIONS OF CEREBELLUM WITH POSTERIOR PETROSAL SURFACE.

IAM, Internal auditory meatus.
SE, Saccus endolymphaticus.
LS, Lateral sinus.
BV, Biventral lobe.

the sigmoid sinus (Fig. 1). The latter crosses obliquely over the lateral margin and anterior part of the under surface of the cerebellum. About three-

quarters of an inch to one inch in front of the middle portion of the sinus, the cerebellar dura is in contact with the internal auditory meatus. Midway between the sinus and the internal auditory meatus the dura is split into two layers which enclose the saccus endolymphaticus.

The middle portion of the under surface of the cerebellum lies above the posterior surfaces of the mid-brain, pons and medulla, and with these structures forms a tent-shaped cavity known as the fourth ventricle. This is continuous above with the aqueduct of Sylvius and below with the central canal of the spinal cord.

The recess on either side of the pons, between it and the under surface of the cerebellum, is known as the cerebello-pontine angle. In this angle are situated the roots of the fifth, sixth, seventh, eighth, ninth and tenth cranial nerves.

The cerebellum is shaped somewhat like a complete clam shell, the hinge of the clam shell lying anteriorly, the edge laterally and posteriorly, and the flattened surfaces above and below. It is divided into a median lobe or vermis, to either side of which is joined a lateral lobe or hemisphere. The vermis alone is present in the lower vertebrates. The lateral lobes appear in the higher forms of vertebrates and attain their maximum development in man.

The upper surface of the cerebellum is slightly

convex, with a low antero-posterior ridge in the center, corresponding to the upper vermis. The lower surface is divided by a deep cleft into two lateral halves. At the bottom of this cleft the lower vermis is seen as

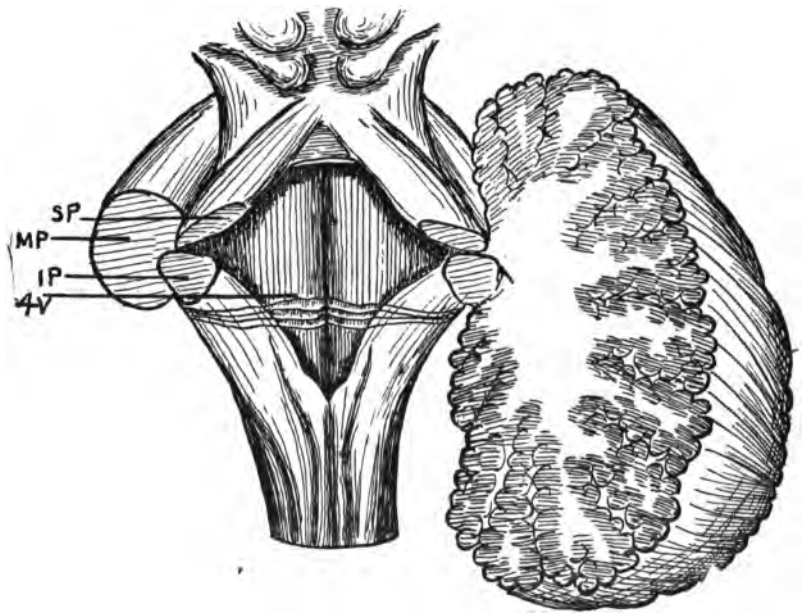


FIG. 2.—CEREBELLAR PEDUNCLES.
SP, Superior peduncle.
MP, Middle peduncle.
IP, Inferior peduncle.
4V, Fourth ventricle.

a well-marked ridge. Into this cleft are received the mid-brain, pons and medulla, to which the cerebellum is attached by means of three pairs of stalks or peduncles, the superior, middle and inferior. (Fig. 2.)

All three peduncles are composed of fibers which

enter into or leave the interior of the cerebellum.

The superior peduncles are two narrow, flat bands which emerge from the upper and mesial part of the hemispheres and pass upward and forward, gradually approaching each other as they ascend. They lie at first in the lateral walls of the fourth ventricle and then in the roof. They disappear from view underneath the posterior corpora quadrigemina.

The middle peduncles are thick bundles of fibers, which emerge from the lateral aspect of the pons and enter the cerebellum just external to the superior peduncles.

The inferior peduncles are continuous with the restiform bodies of the medulla, where they form the lateral walls of the lower part of the fourth ventricle. They enter the cerebellum between the superior and middle peduncles.

The cerebellum is divided into an upper and a lower half by a deep horizontal fissure. The upper half is divided by four sulci into five lobes. (Fig. 3.) The sulci, from before backward, are the precentral, postcentral, preclival and postclival. These fissures divide the upper surface of the cerebellum into lobes which extend transversely across the entire organ. The lobes on the upper surface of the vermis are as follows: In front of the precentral fissure is the lingula; between the pre- and postcentral fissures is the lobus centralis; between the postcentral and pre-

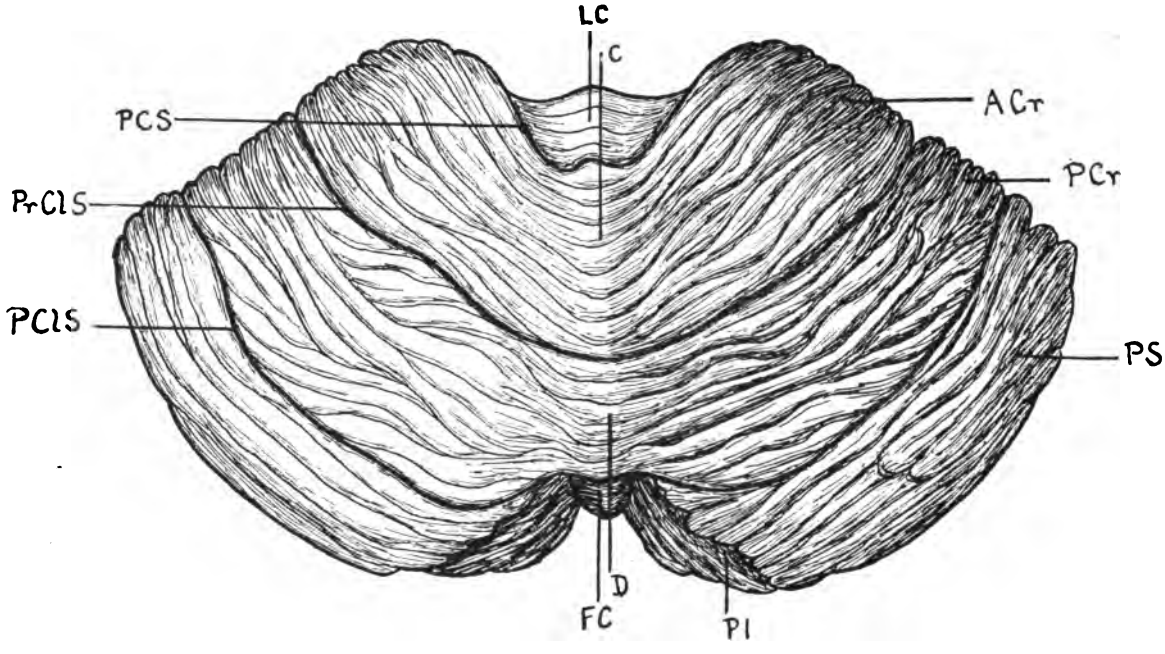


FIG. 3.—CEREBELLUM, FROM ABOVE.

PCS, Postcentral sulcus.
PrClS, Preclival sulcus.
PClS, Postclival sulcus.
LC, Lobus centralis.
C, Culmen.
D, Declive.
FC, Folium cacuminis.
ACr, Anterior crescentic lobe.
PCr, Posterior crescentic lobe.
PS, Postero-superior lobe.
PI, Postero-inferior lobe.

clival fissures is the culmen; between the pre- and postclival fissures is the clivus; and between the postclival and great horizontal fissures is the folium cacuminis.

Every lobe of the vermis is continuous laterally with a lobe in each hemisphere. Thus on either side of the lingula are the frenula; on either side of the lobus centralis are the alæ lobi centralis; on either side of the culmen are the anterior crescentic lobes; on either side of the clivus are the posterior crescentic lobes, and on either side of the folium cacuminis are the postero-superior lobes.

The sulci on the inferior surface of the cerebellum from behind forward are, the post-pyramidal, prepyramidal and post-nodular. (Fig. 4.) The lobes on the under surface of the vermis are as follows: Between the great horizontal and the post-pyramidal fissures is the tuber valvulæ; between the post- and prepyramidal fissures is the pyramid; between the prepyramidal and post-nodular fissures is the uvula, and in front of the post-nodular fissure is the nodulus. The lobes on the under surface of the hemispheres are as follows: On either side of the tuber valvulæ are the postero-inferior lobes; on either side of the pyramid are the biventral lobes; on either side of the uvula are the tonsils, and on either side of the nodule are the flocculi. The biventral lobe is in relation with the posterior surface of the petrous pyramid. The internal

CHAPTER I

ANATOMY OF THE CEREBELLUM

THE cerebellum is situated in the posterior fossa of the skull. It occupies most of that part of the cranial cavity which lies below the tentorium cerebelli. The tentorium is a horizontal process of the dura, attached posteriorly to the inner surface of the occipital bone at the level of the torcular Herophili and the horizontal portions of the lateral sinuses, and laterally to the postero-superior margins of the petrous pyramids. Its anterior margin is crescentic in shape and free, and forms the posterior boundary of an opening for the passage of the mid-brain. It separates the middle from the posterior fossa of the skull. The occipital lobes of the cerebrum rest on its upper surface; the cerebellum is in contact with its lower surface.

The under surface of the cerebellum lies upon that portion of the occipital bone below the groove for the lateral sinus. Its middle portion lies above the mid-brain, pons and medulla. Laterally it is in relation with the posterior surface of the petrous pyramids. Here it is in close proximity to the internal ear. The dura covering this portion of the cerebellum is in

auditory meatus lies against its anterior surface. The descending part of the lateral sinus passes across its posterior portion.

Between the deep fissures, which divide the surface into lobes, there are shallower clefts, which divide the lobes into lobules, so that the surface of the cerebellum has a laminated appearance. The laminæ run transversely.

On section the cerebellum is seen to consist of gray cortex and central white matter. In the white matter are masses of gray substance called the central nuclei. (Fig. 5.) In man there are four of these in each half of the organ, viz.:

1. Nucleus Tecti (nucleus fastigii or nucleus of the roof.)
2. Nucleus Dentatus.
3. Nucleus Emboliformis.
4. Nucleus Globosus.

The vestibular nuclei are often described with the cerebellar nuclei. The latter are para-cerebellar. They are the nucleus of Bechterew, the nucleus of Deiters and the nucleus triangularis. The nucleus emboliformis and the nucleus globosus are considered by Kohnstamm to be upward prolongations of Bechterew's nucleus.

The nuclei tecti are rounded collections of gray matter situated in the white substance of the vermis. They lie just above the roof of the fourth ventricle,

close to the median line, and separated from each other by a thin septum of white matter. They con-

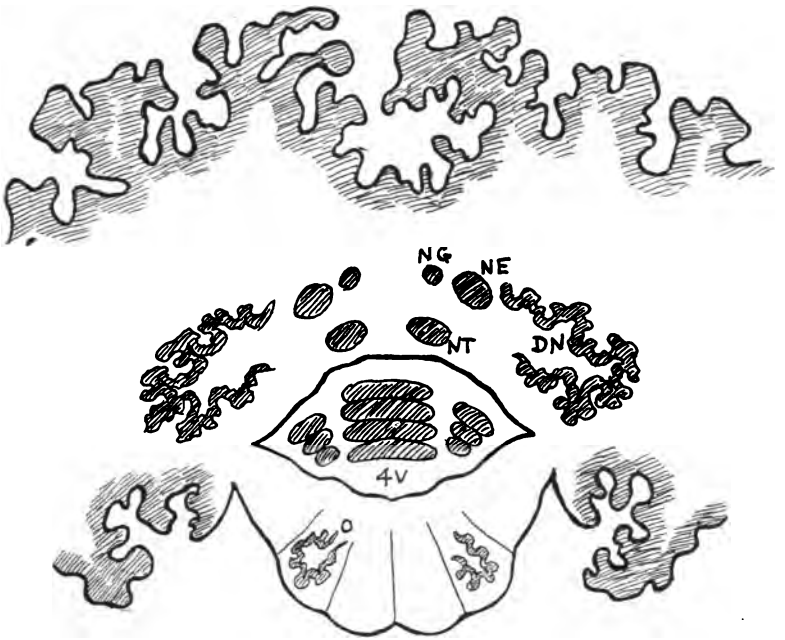


FIG. 5.—VERTICAL TRANSVERSE SECTION THROUGH CEREBELLUM AND MEDULLA.

DN, Dentate nucleus.
NT, Nucleus tecti.
NE, Nucleus emboliformis.
NG, Nucleus globosus.
4V, Fourth ventricle.
O, Medullary olive.

sist of multipolar nerve cells whose axons leave the cerebellum through its peduncles. It is around these cells that the axons of the Purkinje cells terminate.

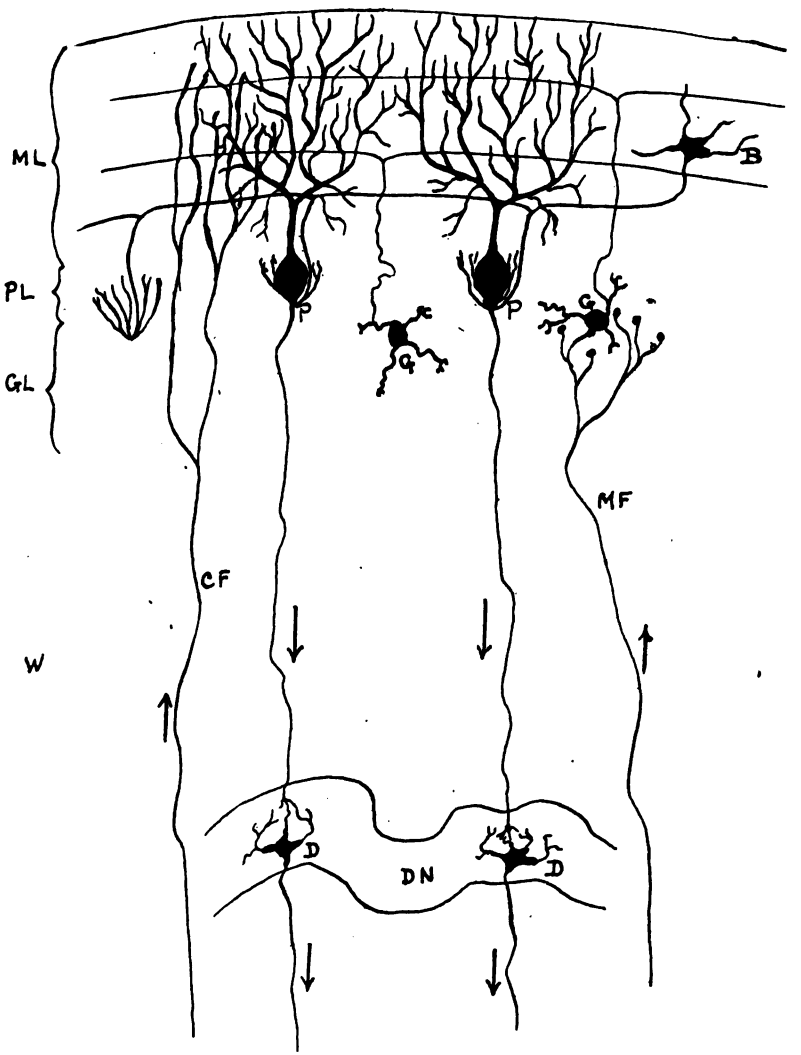


FIG. 6.

FIG. 6.—HISTOLOGICAL STRUCTURE OF CEREBELLUM.

ML, Molecular layer.
PL, Layer of Purkinje cells.
GL, Granular layer.
W, White matter.
DN, Dentate nucleus.
P, Purkinje cell surrounded by network from basket cell.
B, Basket cell.
G, Granule cell.
D, Cells in dentate nucleus.
GF, Climbing fiber or "fibre grimpante."
MF, Mossy fiber.

The nuclei dentati are the largest of the cerebellar nuclei. They are situated near the center of each hemisphere. On section they appear as wavy capsules with an opening situated mesially. This opening is called the hilus. In structure the nuclei dentati are similar to the nuclei tecti.

The nucleus emboliformis lies just mesial to the hilus of the dentate nucleus, and between the nucleus emboliformis and the median line is the nucleus globosus.

The nucleus of Deiters bears a close relationship both physiologically and anatomically to the cerebellar nuclei. It is situated in the dorsal portion of the medulla and pons, between the restiform body and descending root of the fifth nerve. Although, according to Kohnstamm, it receives no fibers directly from the vestibular nerve, yet it receives the vestibular stimuli indirectly through the triangular nucleus. It receives fibers from and sends fibers to the central cerebellar nuclei. It sends fibers through the posterior longitudinal fasciculus to the mid-brain, and through the Deiterso-spinal tract to the anterior horn cells of the cord. The latter tract constitutes one of the important pathways between the cerebellum and spinal cord.

The histology of the cortex of the cerebellum is based upon that of a single lamella, since the structure of the cortex is everywhere the same. Each lamella

is divided from the surface to the interior into four layers (Fig. 6).

1. Molecular layer.
2. Layer of Purkinje cells.
3. Granular layer.
4. White substance.

The outer or molecular layer consists of the dendritic processes of the Purkinje cells, the terminal arborizations of the centripetal cerebellar fibers, neuroglia and two classes of nerve cells, viz.: Small star-shaped cells which lie superficially, and large star-shaped cells (called basket cells) which lie in the deeper portions of this layer. From the body of each large cell an axis-cylinder is given off. This axon, in its course, sends out numerous collaterals, which form basket-like networks about the bodies of the Purkinje cells. The axon itself terminates in a similar manner.

The Purkinje cells lie between the molecular and granular layers. They are large pear-shaped cells whose axons pass down into the white matter and end in the central nuclei of the cerebellum. Their dendritic processes pass outward into the molecular layer like the branches of a tree. The branches spread in the sagittal plane, i. e., at right angles to the direction of the lobules and sulci. These dendritic processes are joined together by the axis-cylinder processes of the cells in the granular layer. The Pur-

kinje cells are thus joined together in two ways, viz.: By the granule cells and by the basket cells.

The granular layer is composed almost entirely of small round cells, which send axons into the molecular layer. Here the axon divides into two branches which run in opposite directions to each other, and at right angles to the axis-cylinder, so that the three form a letter T. The branches run in a coronal plane, i. e., parallel to the cerebellar sulci, and pass across the dendritic processes of the Purkinje cells very much like telegraph wires across the arms of telegraph poles. Thus an impulse from the periphery, originating in a few cells, is transmitted to a large number of the Purkinje cells.

The white substance of the cerebellum is composed of three classes of fibers. These fibers are grouped in accordance with the direction of the impulses which they convey into, (a) association, (b) centripetal, (c) centrifugal fibers.

The association fibers join one lobe or lobule with another, or one hemisphere with the other.

The centripetal fibers enter the cerebellum through the peduncles and terminate in the cortex and central nuclei. Those which end in the cortex are of two types, the so-called mossy fibers and the climbing fibers, or "fibres grimpantes." The mossy fibers have club-shaped extremities and terminate about the granule cells. The climbing fibers end about the

arborizations of the Purkinje cells. Centripetal impulses are therefore transmitted to the Purkinje cells in two ways: Directly, by the climbing fibers and indirectly, by the mossy fibers through the granule cells.

The centrifugal fibers from the cortex consist entirely of the axis-cylinder processes of the Purkinje cells. These all end in the central nuclei, as was proven by Edinger. In numerous experiments he destroyed the cerebellar cortex by means of formalin solution and found, from the degeneration thus caused, that all fibers emanating from the cortex entered the central nuclei, where they stopped. From the cells of the central nuclei centrifugal fibers pass out of the cerebellum through the peduncles.

As has been mentioned above, the cerebellum is connected with the other portions of the central nervous system by its peduncles. In the latter, fiber-tracts pass to and from the cerebellum. The tracts which pass to the cerebellum, i. e., the centripetal tracts, are as follows: (Fig. 7.)

1. Flechsig's tract from the spinal cord through the inferior peduncle to the cortex of the vermis.

2. Gowers' tract from the spinal cord through the superior peduncle to the cortex of the vermis.

3. A tract of fibers from the nuclei of the posterior columns of the cord through the inferior peduncle to the cerebellar cortex.

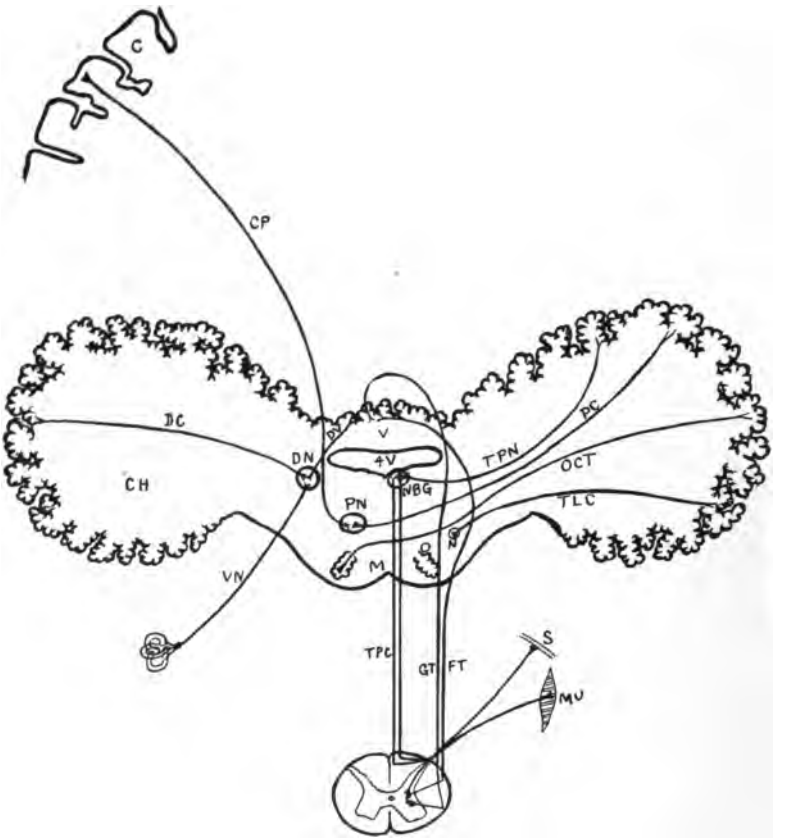


FIG. 7.

FIG. 7.—AFFERENT CEREBELLAR TRACTS.

C, Motor area of cerebral cortex.
CH, Cerebellar hemisphere.
V, Vermis.
M, Medulla.
S, Skin.
MU, Muscle.
CP, Motor tract from cerebral cortex to pontine nuclei.
PN, Pontine nucleus.
PC, Middle peduncle containing fibers from pontine nuclei to opposite cerebellar cortex.
FT, Flechsig's tract.
GT, Gowers' tract.
TPC, Posterior columns of cord.
NBG, Nuclei of Burdach and Goll.
TPN, Tract from nuclei of posterior columns of cord to cerebellum.
N, Nucleus of lateral tract.
TLC, Tract from nucleus of lateral column to cerebellum.
O, Lower olive.
OCT, Olivo-cerebellar tract.
VN, Vestibular nerve.
DN, Deiters' nucleus.
DC, Fibers from Deiters' nucleus to cerebellar hemisphere.
DV, Fibers from Deiters' nucleus to vermis.
4V, Fourth ventricle.

4. A tract from the nucleus of the lateral columns in the medulla through the inferior peduncle to the cerebellar cortex.

5. Olivo-cerebellar fibers.

6. Tracts from Deiters' nucleus to the cerebellar cortex and central nuclei.

7. Tracts from the motor cerebral cortex through the middle peduncle to the opposite cerebellar hemisphere.

The tracts which pass from the cerebellum, i. e., the centrifugal tracts, are as follows: (Fig. 8.)

1. The tractus uncinatus from the nucleus tecti to Deiters' nucleus of the opposite side. From here a secondary tract—the Deiterso-spinal tract—passes down in the anterior column of the cord and ends about the anterior horn cells.

2. A tract from the dentate nucleus to Deiters' nucleus.

3. Tracts from the central nuclei of the cerebellum to the nuclei of the posterior columns of the cord.

4. Tracts from the central nuclei of the cerebellum to the nuclei of the lateral column.

5. The brachium conjunctivum and accessory brachium from the dentate nucleus and nucleus tecti through the superior peduncle to the opposite red nucleus and optic thalamus. From the red nucleus a secondary tract—the rubrospinal tract—passes

down in the opposite lateral column of the cord and terminates about the anterior horn cells.

6. The ventral cerebello-thalamic bundle, from the dentate nucleus to the opposite corpora quadrigemina and optic thalamus.

Flechsig's tract (direct cerebellar tract; dorsal spino-cerebellar tract) begins in the lower lumbar region of the cord. It arises from a group of cells (Clarke's column) which is situated at the base of the posterior horn. The fibers of this tract are collected into a bundle which is situated at the posterior part of the margin of the lateral column of the cord, behind Gowers' tract. At the upper end of the cord it lies next to the substantia gelatinosa, i. e., the head of the posterior cornu. As it passes into the medulla it is separated from this by the spinal root of the fifth nerve and then enters the corpus restiforme or inferior peduncle. It passes through the peduncle to the cerebellum where its fibers are distributed to the cells in the cortex of the upper worm of both sides.

Gowers' tract (ventral spino-cerebellar tract) takes its origin in a column of cells situated in the lateral part of the base of the anterior horn and central gray substance. The tract begins in the lower lumbar region. It is situated at the anterior portion of the margin of the lateral column of the cord in front of Flechsig's tract. At the upper end of the cord it separates from Flechsig's tract, passing ventrally to the

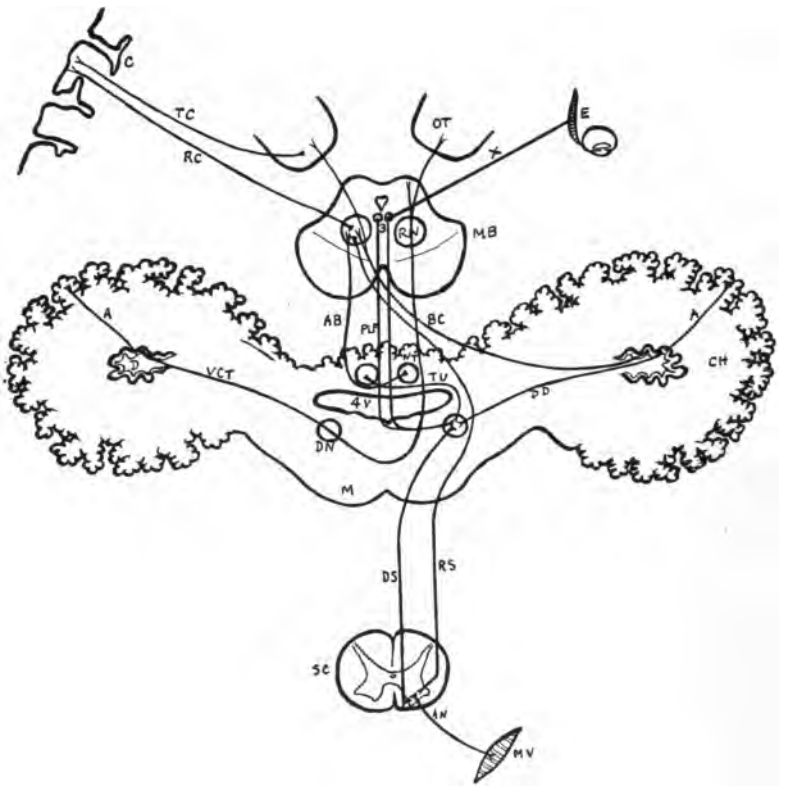


FIG. 8.

FIG. 8.—EFFERENT CEREBELLAR TRACTS.

C, Cerebral cortex.
MB, Mid-brain.
CH, Cerebellar hemisphere.
M, Medulla.
SC, Spinal cord.
E, Ocular muscle.
MV, Muscles of trunk and extremities.
OT, Optic thalamus.
RN, Red nucleus.
3, Oculo-motor nuclei.
NT, Nucleus tecti.
D, Dentate nucleus.
DN, Deiters' nucleus.
4V, Fourth ventricle.
A, Fibers from cortex of cerebellum to central nuclei.
BC, Brachium conjunctivum.
RC, Fibers from red nucleus to cerebral cortex.
TC, Fibers from optic thalamus to cerebral cortex.
RS, Rubro-spinal or Monakow's tract.
AB, Accessory brachium conjunctivum.
VCT, Ventral cerebello-thalamic bundle.
DD, Tract from dentate to Deiters' nucleus.
TU, Tractus uncinatus.
DS, Deiterso-spinal tract.
AN, Anterior nerve root.
PLF, Posterior longitudinal fasciculus.
X, Oculo-motor nerve.

spinal root of the fifth nerve and reaches the anterior outer portion of the medulla. It passes between the nucleus of the facial nerve and the upper olive and then along the outer side of the lateral fillet to the anterior extremity of the pons. Here it winds around the brachium conjunctivum, reaches its inner side, and, turning back along with the brachium, it enters the cerebellum. Its fibers end in the cortex of the worm of both sides.

Fibers originating in the nuclei of the posterior columns of the cord, i. e., the nuclei of Goll and Burdach, pass through the inferior peduncles to the cortex of the cerebellar hemispheres. Some of the fibers of the columns of Goll and Burdach enter the cerebellum directly, without being interrupted by the nuclei of these columns.

A tract from the nucleus of the lateral column in the medulla passes through the inferior peduncle to the cerebellar cortex.

From the inferior olive internal arcuate fibers pass through the opposite restiform body to the cortex of the cerebellar hemisphere.

Fiber tracts pass from Deiters' nucleus to the cortex and central nuclei of the cerebellum. These are in all probability part of the vestibulo-cerebellar tracts.

From the cortex of the cerebral hemisphere fibers pass through the internal capsule and crus cerebri and

terminate about cells scattered through the anterior portion of the pons. From these cells axons cross in the pons and, passing through the middle peduncle, end in the cortex of the opposite hemisphere.

The fibers of the tractus uncinatus arise in the nucleus tecti; they cross over in the roof of the fourth ventricle to the opposite cerebellar hemisphere, where they pass into Deiters' nucleus.

A tract of fibers passes from the dentate nucleus to Deiters' nucleus of the same side. Fibers also pass from the dentate nucleus to the nuclei of the posterior columns and to the nucleus of the lateral column.

The fibers of the brachium conjunctivum (Fig. 9) arise in the dentate nucleus of the cerebellum and cross over through the tegmentum of the pons to the tegmentum of the opposite side of the mid-brain where most of them end in the red nucleus. A few pass to the optic thalamus.

An accessory brachium conjunctivum arises from the nucleus tecti and crosses to the opposite side of the vermis where the fibers form a sort of hood covering the brachium. These fibers also end in the opposite red nucleus and optic thalamus.

From the red nucleus and optic thalamus fibers pass up through the internal capsule to the sensori-motor area of the cerebral cortex. From the red nucleus a fiber tract also passes downward through the tegmentum of the pons and medulla to the opposite lat-

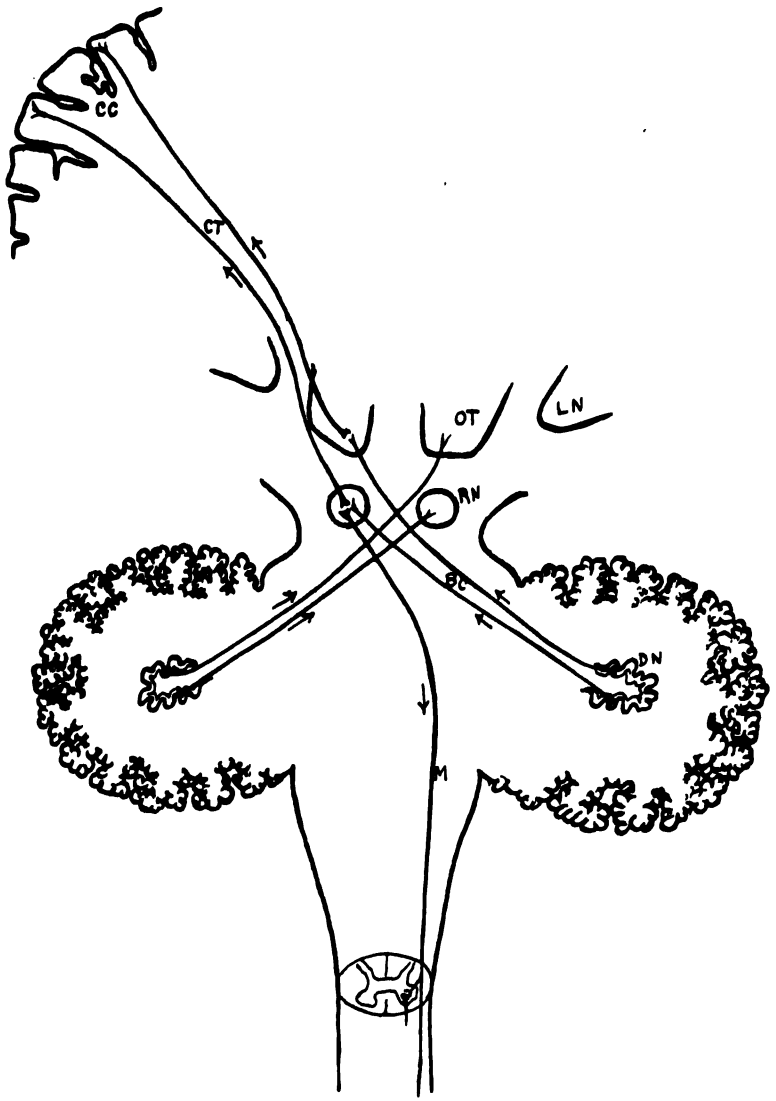


FIG. 9.

FIG. 9.—BRACHIUM CONJUNCTIVUM AND SECONDARY TRACTS FROM RED NUCLEUS AND OPTIC THALAMUS.

DN, Dentate nucleus.
RN, Red nucleus.
OT, Optic thalamus.
LN, Lenticular nucleus.
CC, Cerebral cortex.
BC, Brachium conjunctivum.
M, Monakow's tract or rubro-spinal tract.
CT, Tract from red nucleus and optic thalamus to sensori-motor area of cerebral cortex.

eral column of the spinal cord. It passes down through the entire length of the spinal cord, lying in front of the crossed pyramidal tract and gives off collaterals which terminate about the anterior horn cells. This tract is called the rubro-spinal or Monakow's tract. (Fig. 10.) In this way each cerebellar

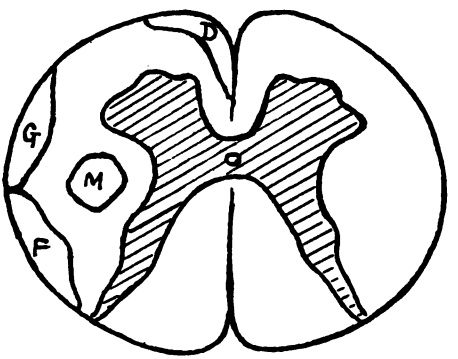


FIG. 10.—SPINO-CEREBELLAR TRACTS.
M, Monakow's or rubro-spinal tract.
F, Flechsig's tract.
G, Gowers' tract.
D, Deiterso-spinal tract.

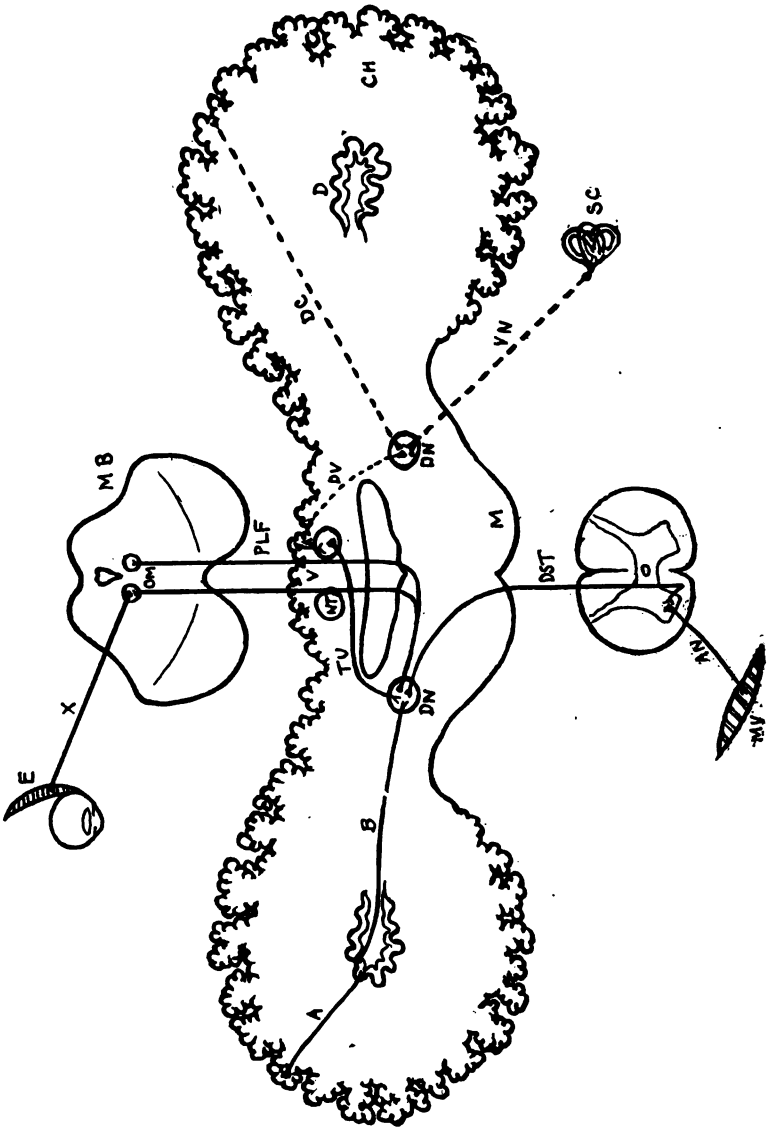
hemisphere is connected with the opposite cerebral hemisphere and with the anterior horn cells of the same side of the cord.

The ventral cerebello-thalamic bundle takes origin, according to Probst, in the dentate nucleus. The fibers pass through Deiters' nucleus and then as inner arcuate fibers they reach the central portion of the substantia reticularis of the pons. Here they cross the median raphé, bending around into the sagittal

plane between the raphé and mesial fillet. They then pass upward and terminate in the corpora quadrigemina and the inner portion of the optic thalamus.

According to Kohnstamm, the nucleus of Deiters does not receive the fibers of the vestibular nerve directly, but receives the vestibular impulses through the intermediation of the triangular nucleus. It receives the tractus uncinatus from the nucleus tecti and probably some fibers from the dentate nucleus. (Fig. 11.) It sends out two centrifugal tracts; one to the spinal cord and the other to the posterior longitudinal fasciculus. The fibers which pass down into the cord are known as the Deiterso-spinal tract. They pass inward from Deiters' nucleus as inner arcuate fibers to the raphé, where they bend downward forming a bundle at the margin of the anterior fissure of the cord and the adjacent portion of its ventral border. Passing down to the sacral portion of the cord they give off in their course collaterals to the anterior horn cells.

From Deiters' nucleus arcuate fibers pass to the posterior longitudinal fasciculus of both sides. These fibers end about the cells of the nuclei of the third, fourth, and sixth nerves.


CH
D
SC
DC
VN
DN
dV
MB
M
PLF
DST
Om
V
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X
E
A
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AV

FIG. 11.—TRACTS TO AND FROM DEITERS' NUCLEUS.

(Afferent tracts to cerebellar cortex are indicated with dotted lines and efferent tracts from cerebellar cortex with solid lines.)

MB, Mid-brain.
CH, Cerebellar hemisphere.
V, Vermis.
M, Medulla.
SC, Semicircular canals.
E, Ocular muscle.
MV, Muscles of trunk and extremities.
OM, Oculo-motor nuclei.
D, Dentate nucleus.
NT, Nucleus tecti.
DN, Dentate nucleus.
VN, Vestibular nerve.
DC, Tract from Deiters' nucleus to cortex of cerebellar hemisphere.
DV, Tract from Deiters' nucleus to cortex of vermis.
A, Fibers from cerebellar cortex to central nuclei.
B, Fibers from dentate nucleus to Deiters' nucleus.
TU, Tractus uncinatus.
DST, Deiterso-spinal tract.
AN, Anterior nerve root.
PLF, Posterior longitudinal fasciculus.
X, Oculo-motor nerve.

CHAPTER II

PHYSIOLOGY OF THE CEREBELLUM

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PHYSIOLOGY OF THE CEREBELLUM

DESPITE the fact that for many years both the structure and the function of the cerebellum have been the subject of a great deal of valuable research, there is still much concerning the physiology of this organ that is unknown. Of the experimental methods employed for the study of cerebellar function, three stand out prominently. The first is the destruction or removal of certain parts of or all of the organ and section of its afferent or efferent tracts; the second is the stimulation, by means of the electric current, of the cerebellar cortex and the central nuclei; and the third is the freezing of portions of the cerebellar cortex in man.

The great difficulty encountered by most of those who experimented with the first two methods, lay in the fact that a multitude of centers and fiber pathways were thus destroyed or stimulated at one time. Moreover, the proximity of extraneous centers and nerve pathways to the field of operation rendered injury to or stimulation of these almost unavoidable, so that the phenomena following such procedures could not be classified or interpreted. After removal

of parts of the cerebellum it was impossible to differentiate between phenomena that resulted from destruction and those which resulted from irritation. Nor was it possible to determine which reactions were compensatory and which were due to irritation or destruction of neighboring parts.

The result of this confusion was that, although many experimenters observed similar phenomena following destruction of parts of the cerebellum, the interpretations of these phenomena varied and there was no unanimity of opinion regarding the functions ascribed to this organ.

According to Lewandowsky, the phenomena following removal of half the cerebellum in the dog group themselves into two periods. The first is the period of forced movements (Zwangsbewegungen); the second is the period of incoördination. The forced movements consist chiefly of a curving of the body, a torsion of the neck and an extension of the extremities. They occur spontaneously, but are more marked when the animal attempts to execute any voluntary movement. The forced movements are, of course, caused by muscular action, yet there is neither paralysis nor spasticity of either side of the body. It follows, then, that these symptoms are not simple motor phenomena such as would follow, for instance, unilateral section of the spinal cord. They result from a peculiar combination of muscular activity and this

combination is, in all probability, the province of the cerebellum.

The cerebellum sends out a continuous stream of impulses which result in a condition of tonicity of the body musculature. Following a lesion in one cerebellar hemisphere the impulses from the normal half predominate, and thus produce the forced movements.

As the period of forced movements comes to a close and the animal makes efforts to stand and to walk, it becomes apparent that there is marked difficulty in holding the body erect while standing and in maintaining equilibrium during progression. This is the period of incoördination. The cerebellum is therefore an important organ in the process of equilibration and its destruction experimentally in animals or by disease in man entails a serious disturbance of body balance.

According to Lewandowsky equilibration results from the combined activities of the muscles of the trunk, head, eyes, and extremities. It consists of maintenance of the body erect in space against the force of gravity and in the possibility of holding a certain direction of movement. It stands in close relationship to the ability to compensate for passive rotation of the body and head about their axis.

If, then, we consider the phenomena following lesions of the cerebellum as disturbances of the orienta-

tion of the body, we have confronting us the questions:

1. Through which peripheral organs and by what centripetal pathways are the factors of such orientation carried to the cerebellum?

2. What processes are aroused in the cerebellum?

3. Through what centrifugal pathways are these impulses carried from the cerebellum, and,

4. What part does the cerebrum play in the performance of this function of orientation?

Equilibration is the result partly of conscious impressions and partly of reflex activities. The reflex element is probably the more important of the two. The conscious elements which enter into the maintenance of equilibrium are vision, touch, and to a lesser extent hearing and smell. While our knowledge of circumferential space is gained, to a certain extent, from these sources, it is locomotion which really enables us to learn the details of space and it is by the translation of our bodies and limbs that we experience and remember the depths and distances that separate us from any definite point in any plane of the space surrounding us. Our knowledge of movements of the head is obtained through the intermediation of a special organ of orientation, viz., the static labyrinth.

As the result of the information received from these various conscious impressions we perform voluntary

movements in order to place our bodies in certain positions and to move about in space. These voluntary movements result from impulses which arise from the motor area of the cerebral cortex and pass through the internal capsule and pyramidal tracts to the anterior horn cells of the cord. From here they pass through the anterior nerve roots to the muscles of the head, trunk and extremities.

But much more important than these conscious impressions and voluntary impulses in the performance of orderly movements and the preservation of body balance are the reflex activities of the cerebellum. If we consider the important peripheral sources from which knowledge of our spatial relations is derived we find that they consist of the skin, muscles, joints and the static labyrinth. Without doubt, the recorded memory of space, so far as knowledge is attained by locomotion, by exploratory movements of our limbs, by our sense of touch and from the static labyrinth, finds its physical location in the kinesthetic area of the cerebral cortex. Across the nerve pathways, in a sense, which transmit impulses from these peripheral sources to the cerebral cortex the cerebellum stands as the center of a reflex arc. To it are conveyed impulses from the skin, muscles, joints and static labyrinth, and from it emanate impulses which reflexly establish and maintain equilibrium.

From the muscles, articular surfaces, ligaments and tendons impulses are constantly being transmitted to the cerebellar cortex through the posterior nerve roots and Flechsig's and Gowers' tracts. These impulses arise even when the body is at rest. From the skin impulses are sent to the cerebellar cortex through the posterior nerve roots and the columns of Goll and Burdach. From the static labyrinth impulses are sent to the cerebellum through the vestibular nerves and Deiters' and Bechterew's nuclei. (Fig. 12.)

These afferent impulses are gathered together in the cerebellar cortex and are projected along the axons of the Purkinje cells to the central nuclei. In the latter secondary impulses arise which are transmitted to the so-called nucleus motorius tegmenti. This is composed of a column of cells in the tegmentum of the mid-brain, pons and medulla, of which the principal enlargements are situated at the red nucleus in the mid-brain and Deiters' nucleus in the pons. From the cerebellar nuclei impulses reach the opposite red nucleus via the brachium conjunctivum. They reach the opposite nucleus of Deiters' by way of the tractus uncinatus. From the red nucleus the impulses, crossing again, pass through the rubrospinal or Monakow's tract to the anterior horn cells. From Deiters' nucleus they pass through the Deiterso-spinal tract to the anterior horn cells. Through the anterior nerve roots the impulses reach the muscles. Thus the

reflex arc is completed. As these centrifugal tracts cross twice between the cerebellar hemisphere and the anterior horn cells the effect is homolateral.

With regard to the question as to what processes are aroused in the cerebellum as the result of the affer-

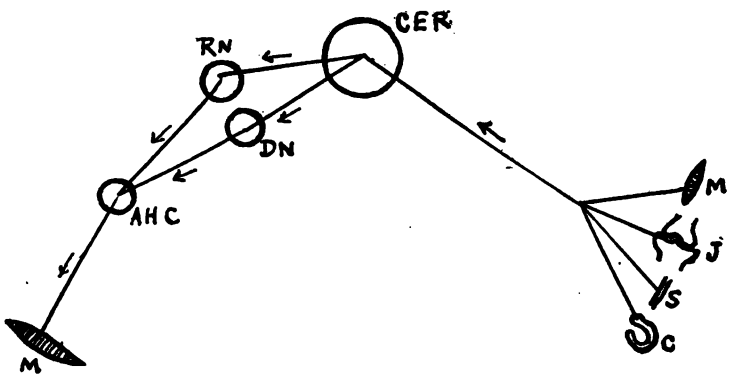

FIG. 12.—CEREBELLAR REFLEX ARC.

M, Muscles.	CER, Cerebellum.
J, Joints.	RN, Red nucleus.
S, Skin.	DN, Deiters' nucleus.
C, Semicircular canals.	AHC, Anterior horn cell.

ent impulses, there is no doubt that the cerebellum originates motor impulses. That these impulses are independent of the cerebrum has been shown often in rabbits in which the cerebrum was removed in toto and then the cerebellum exposed and irritated electrically. In dogs too, after removal of the motor centers of the extremities in the cerebrum, cerebellar irritation was

followed by motor effects. As was pointed out in discussing forced movements, the cerebellar processes which result from afferent impulses are not simple motor phenomena.

The various centripetal impulses from muscles, joints, skin and static labyrinth result in a continuous stream of centrifugal impulses which produce a certain tonus in the muscles of the body. The purpose of this muscular tonus or tension is to preserve the body balance. The tension in the muscles of one side of the body counterbalances that in the muscles of the opposite side and thus equilibrium is preserved. As the muscles and joints are moved and as the position of the head is changed, the various centripetal impulses change and result in corresponding changes in the centrifugal portion of the reflex arc. In this way the tension in the muscles of different parts of the body is altered to meet the new conditions.

When there is a lesion in one half of the cerebellum the impulses on that side are diminished and those from the opposite side overbalance. The result is a disturbance of equilibrium and the production of the so-called forced movements. When these disturbances occur in the trunk muscles they result in falling. When they occur in the muscles of the extremities they result in deviations of the extremities or the so-called "pointing by." These disturbances of equilibrium may result from abnormalities in the centripetal

impulses as well as from lesions in the cerebellum itself, as, for instance, in disease of the static labyrinth or vestibular nerve or from lesions of the posterior nerve roots of the spinal cord. They may also occur from abnormally strong centripetal impulses such as are aroused by the rotation or caloric tests.

Horsley and Clark have shown, by their experiments on the electrical stimulation of the cerebellum, that the closer the current is applied to the central nuclei, the greater is the reaction. The conclusions which they draw correspond with Edinger's view, viz.: That the cerebellar cortex is purely sensory. Rothmann and Shimazono, on the contrary, believe that they have aroused motor phenomena from stimulation of the cerebellar cortex alone. Probably these views differ apparently more than really. Rothmann himself in discussing the division of the brachium and the accessory brachium at the red nucleus states: "They probably convey the same impulse. The former through the rubrospinal tract, however, results in motion earlier than the latter which passes to the thalamus and so to the cerebral cortex." There is then, in all probability, no great essential difference between the impulses which the cerebellar cortex originates and those which emanate from its central nuclei.

Edinger divides the cerebellum on physiological as well as anatomical grounds into a paleo- and neo-

cerebellum. To the paleo-cerebellum, i. e., the vermis, he ascribes the principal part in the development of what he calls the statotonus, i. e., such tonic influences over the muscles as enable them to maintain the body erect while standing and during progression.

To the experiments of Bing we owe our knowledge of the fact that the afferent impulses from the muscles, joints and tendons which arouse the statotonus in the vermis pass to the latter through the spino-cerebellar tracts. These tracts, as is well known, end almost exclusively in the cortex of the vermis. Bing, pointing out the danger of ambiguity and confusion in the interpretation of the symptom-complex following surgical experiments upon the cerebellum itself, urged the reduction of the problem of cerebellar physiology to it utmost simplicity. Following this dictum he destroyed the continuity of Flechsig's and Gowers' tracts in the cord and observed, as a sequence, a marked disturbance in the statotonus.

With this view of the physiological activity of the vermis Barany also agrees, and he ascribes the reaction movements of the body following vestibular impulses to the intermediation of the cortex of the vermis. Neither pathological nor experimental proof, however, has as yet fully confirmed this theory.

Rothmann, in discussing Edinger's theory, holds that the statotonic mechanism is solely the province of the paleo-cerebellum, i. e., the vermis. With the

development of the neocerebellum, i. e., the lateral hemispheres, in the higher mammals, there occurs in the cerebellum a mechanism for the control of movements of the extremities in various directions. Clinically we have long known that one of the most valuable signs of cerebellar disease is the loss of power to carry the hand to any definite point in a coördinate manner. In the study of this phenomenon and the effect of vestibular impulses upon movements of the limbs Barany has evolved a theory of cerebellar localization.

He believes that the various body muscles are not represented in the cerebellar cortex as they are in the cerebral cortex, but that in the former the muscles are grouped into direction centers. Within these centers there is a subdivision according to joints and according to the position of joints. In each half of the cerebellum there are four direction centers; one for movement upwards, one for movement downwards, one for movement to the right and one for movement to the left. In the center for movement to the right there are centers for the shoulder, wrist and hip joints, etc., and in the center for the wrist joint there is a center for movement with the hand in pronation and one for movement with the hand in supination.

When there is a lesion, for example, in the center for movement inward at the shoulder joint, the arm of that side will deviate outward. This deviation can

be elicited only during voluntary motion, as when the arm is held extended, or when an attempt is made to point to some object. The deviation is most marked when the eyes are closed, for if the patient becomes aware of his deviation he makes a voluntary effort to correct it. The deviation is greatest after sudden destruction of a center. After a certain time it may disappear as the result of compensation, some other part of the cerebellum or some other part of the central nervous system taking up the function of the destroyed area.

Disturbance of the centripetal impulses such as occurs with stimulation or destruction of a static labyrinth will give rise to deviations which are very similar to those produced by destruction of the cerebellar centers.

When, as a result of stimulation of the semicircular canals in an individual with a normal cerebellum, a labyrinthine nystagmus is aroused, there will occur a deviation of all four extremities in the plane of the nystagmus and in the direction of its slow component. This is called the pointing reaction. But when there is a cerebellar lesion, the extremity effected by the lesion will point correctly in the presence of a vestibular nystagmus. In other words there is a loss of the pointing reaction. A lesion in one cerebellar hemisphere causes a deviation and a loss of the pointing reaction only in the extremities of the same side.

For example, with a lesion in the right cerebellar hemisphere involving the center for movement to the left of the shoulder joint, the right arm extended in front of the body would deviate to the right; the left arm would point correctly. If, now, a horizontal nystagmus to the right is produced either by cold irrigation of the left ear or by rotation to the left, the right arm would point correctly whereas the left arm, which received the stimuli from the intact cerebellar hemisphere, would deviate to the left.

When the lesion of the cortex is a large one many joints will be involved in the deviation; when the lesion is small only one or two joints will be involved.

If a caloric test is used to arouse the reaction movements, the head should be tilted backward about sixty degrees in order to elicit a horizontal nystagmus. In this way lateral deviations of the extremities are produced. If a cold caloric is done in the left ear and the head held upright, a mixed rotatory and horizontal nystagmus to the right will occur. If the arms are held in front of the body, they will deviate to the left. If the right arm is extended laterally, it will deviate upward. If the left arm is extended laterally, it will deviate downward. If the head be now rotated to the right shoulder, both arms held in front of the body will deviate downward. If the head be rotated to the left shoulder, both arms held in front of the body will deviate upward. It is clear, from these facts, that

the deviations are dependent upon impulses which arise from the static labyrinth and from the muscles and joints of the neck. These impulses are mixed, as it were, in the cerebellar cortex and there arouse processes which influence the voluntary impulses from the cerebrum. In this way the reaction movements or deviations occur. The deviation is always in the plane of the nystagmus and in the direction of the slow component of the latter.

When there is spontaneous deviation but no loss of reaction movement in response to vestibular stimulation, the lesion is probably remote and causes impairment of function by pressure and not by actual destruction of the center.

A deviation of the right arm to the right may be due either to destruction of the right center for inward tonus of the shoulder joint, or to hyperstimulation of the right center for outward tonus of the shoulder joint. In order to determine which of these conditions is present, the reaction movements of the right arm should be compared with those of the left arm. If the reaction movement to the left of the right arm is wanting, there is destruction of the right center for inward tonus. If the reaction movement to the right of the right arm is greater than that of the left arm, there is hyperexcitability of the right center for outward tonus of the shoulder.

When the deviation is in one direction only and

with increase in intracranial pressure it spreads to all joints without taking a new direction there is, in all probability, a cortical cerebellar lesion. This is analogous to monoplegia resulting from a lesion in the cerebral cortex. When the deviation and the loss of reaction movements is in more than one direction there is probably a lesion in the central portion of the cerebellum involving the fiber-tracts.

Lesions of the cerebellar hemispheres cause deviations of the extremities. Lesions of the central lobe or vermis cause disturbances of the trunk muscles with loss of equilibrium. If an artificial rotatory nystagmus is aroused by means of cold or hot water in the ear, the patient falls in the direction of the slow component of the nystagmus when the vermis is intact. For example, if a cold caloric is done in the right ear the patient falls to the right. If the head be turned to the left shoulder he falls forward. If the head be turned to the right shoulder he falls backward. As the reaction movements of the extremities, so the reaction movements of the trunk are dependent upon impulses from the static labyrinth and from the muscles and joints of the neck. They are always in the plane of the nystagmus and in the direction of the slow component of the latter.

If there is a lesion of the vermis, there is spontaneous falling, usually toward the side of the lesion. The direction of this spontaneous falling will not be

changed by the production of a caloric vestibular reaction. In other words, the "falling reaction" is wanting. The loss of the falling reaction may be in one

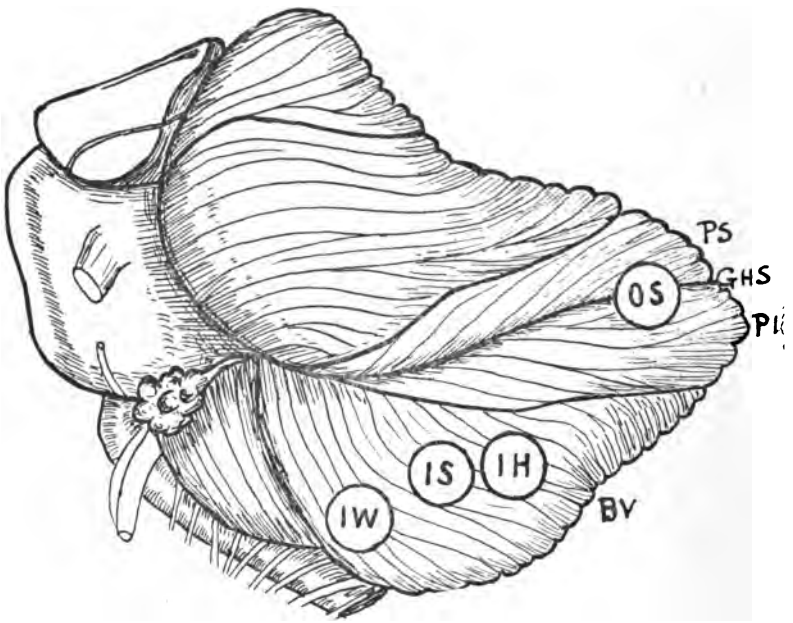

FIG. 13.—CEREBELLUM, LATERAL VIEW.
IW, Center for inward tonus of wrist.
IS, Center for inward tonus of shoulder.
IH, Center for inward tonus of hip.
OS, Center for outward tonus of shoulder.
PS, Postero-superior lobe.
PI, Postero-inferior lobe.
GHS, Great horizontal fissure.
BV, Biventral lobe.

or several directions. In the latter case the lesion would be larger or more centrally located than in the former case.

Whether we agree with Barany's conclusions or not, we cannot deny the results of his experiments of freezing parts of the cerebellar cortex, for they, too, reduce the problem of cerebellar physiology to its elements.

By means of freezing portions of the cerebellar cortex as well as through operative and pathological lesions of the cerebellum, Barany was able to map out definite tonus centers. He found that there is a region in the cortex of the biventral lobe, just behind that portion of the cerebellum which lies against the internal auditory meatus, which controls inward movement of the wrist joint. (Fig. 13.) The center for position with the palm downward is located in the medial portion of this area and that for position with the palm upward in the lateral portion. A little behind and above this area is the center which controls inward movement of the shoulder joint, and just behind this, the center which controls inward movement of the hip joint.

By freezing the shoulder area he has repeatedly shown that there follows a spontaneous deviation of the arm of the affected side outward and a loss of the pointing reaction of that arm inward. In twenty cases of pathological and operative lesions of this area the same phenomena were observed. Near the posterior pole of the cerebellar hemisphere, viz., at the medial end of the postero-superior and postero-

inferior lobes, is the center for downward movement of the shoulder joint, and three to four centimeters outward from this point, at the edge of the hemisphere, viz., at the middle of the postero-superior and postero-inferior lobes, is the center for outward movement of the shoulder joint. (Fig. 14.)

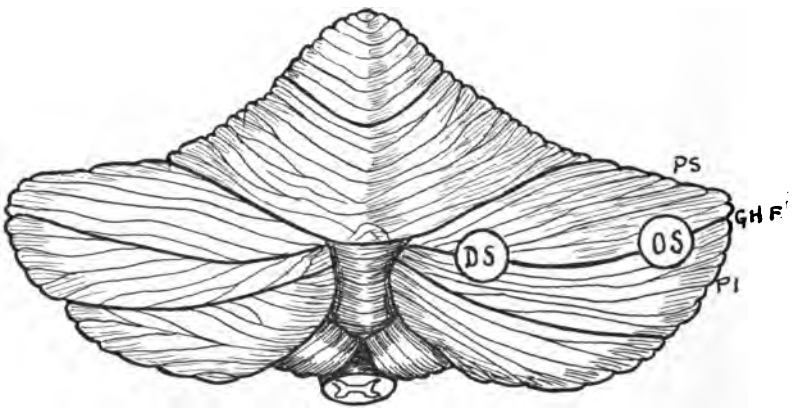

FIG. 14.—CEREBELLUM, POSTERIOR VIEW.
DS, Center for inward tonus of shoulder.
OS, Center for outward tonus of shoulder.
PS, Postero-superior lobe.
GHF, Great horizontal fissure.
PI, Postero-inferior lobe.

We come now to the fourth question which we have propounded for ourselves, viz., what part does the cerebrum play in this function of static orientation? There is no question that conscious impulses enter into the maintenance of body balance, for during unconsciousness equilibrium is abolished. Yet the processes which serve this purpose are carried on in the subcon-

scious plane. The voluntary impulses which are used for maintaining equilibrium and for controlling the movements of the extremities pass into the cerebellum via the middle cerebellar peduncles. In the cerebellum they meet the centripetal impulses from the muscles, joints, skin and static labyrinth. These centripetal impulses modify the motor cerebral impulses in such a way as to make them efficacious in preserving body balance. The cerebrum originates the movements and the cerebellum carries them out.

The cerebellum exercises an inhibitory control over the vestibular centers and also over the motor areas in the cerebral cortex. That it exercises an inhibitory control over the vestibular centers is shown by the following fact. If with a normal cerebellum a cold caloric is done and the flow stopped as soon as nystagmus sets in, the nystagmus will continue for one or two minutes. In cerebellar disease, the caloric nystagmus which is directed toward the affected hemisphere will continue for five or ten minutes or even longer and is much more intense than usual. This has been called, by Neumann, "enduring nystagmus." That the cerebellum exercises an inhibitory control over the motor area in the cerebral cortex was shown by I. L. Meyers in the following ingenious way: Excited tissue is electrically negative in relation to quiescent tissue. The galvanic current travels from the quiescent to the active part, i. e., from the positive to

the negative pole in the external circuit. In the tissue portion of the circuit it travels from the active to the quiescent part. When half of the cerebellum was removed, in a cat, and both sciatic nerves exposed and joined together by means of a wire, in the course of which a galvanometer was interposed, the deflection of the galvanometer needle showed that the nerve on the side of the cerebellar lesion was negative, and the one on the sound side positive. In other words, the nerve on the side of the lesion was in a state of hyperexcitability. When the contralateral motor area in the cerebrum was removed the galvanometer deflection ceased. This proves that the cerebellar hemisphere exercises an inhibitory control over the opposite motor area in the cerebrum. But that the cerebellum has motor functions independent of the cerebrum was shown by Rothmann, who removed the cerebrum in rabbits and was still able to produce muscular contraction by electrical stimulation of the cerebellum.

That the cerebellum has a tonic influence on the musculature of the body is shown by the hypotony and atony that ensue from cerebellar disease and from experimental lesions of the cerebellum in animals.

The various disturbances which occur as a result of lesions of the cerebellum are most marked immediately after the lesion has occurred. They gradually diminish in intensity and in some cases entirely dis-

appear in time. This is due to the fact that the function of the injured portion of the cerebellum is taken up either by some other part of the cerebellum or by some other part of the central nervous system. When the lesion in the cerebellum is produced very slowly there may be no symptoms whatever, compensation keeping pace with the destruction. In the cases of animals with cerebellar lesions who have regained their equilibrium, destruction of the motor cerebral cortex causes a recurrence of the loss of equilibrium. That other portions of the cerebellum compensate for the destroyed portions is proven by the following fact: If one inferior peduncle is destroyed in a dog, there occur rolling movements around the antero-posterior axis of the body toward the operated side. If the other inferior peduncle is then divided, the rolling movements cease and symptoms of static incoördination appear. If, however, after section of the first side, sufficient time is allowed to elapse for compensation to take place, and then the second side is divided, there occur rolling movements toward the newly operated side.

CHAPTER III

ETIOLOGY AND PATHOLOGY OF CEREBELLAR ABSCESS

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ETIOLOGY AND PATHOLOGY OF CEREBELLAR ABSCESS

CEREBELLAR abscess is, in the vast majority of instances, otitic in origin. Of eighty-six cases of cerebellar abscess collected from the literature since 1907, eighty-five were due to suppuration within the temporal bone. The remaining case was traumatic in origin. Among other causes may be mentioned abscess or gangrene of the lung, purulent bronchitis, empyema, purulent pericarditis, compound fractures, pyemia and tuberculosis.

Cerebellar abscess may complicate either an acute or a chronic suppuration of the middle ear and mastoid. It is, however, far more frequently associated with the chronic otitides. In the eighty-six cases collected, fourteen (16.2 per cent) complicated the acute otitides, while seventy-two (83.8 per cent) followed the chronic suppurations. Okada found acute middle-ear suppuration as the causative factor in twenty-eight cases (19 per cent) of cerebellar abscess, and chronic middle-ear suppuration in one hundred and twenty cases (81 per cent).

Heimann found 20 per cent following acute mid-

dle-ear suppuration and 80 per cent following chronic suppuration. Neumann found 12 per cent following acute suppuration and 88 per cent following chronic suppuration.

Grunert found 9 per cent following acute suppuration and 91 per cent following chronic suppuration.

Hammerschlag found 25 per cent following acute suppuration and 75 per cent following chronic suppuration.

The reason for this undoubtedly lies in the fact that in the chronic cases, there is greater destruction of bone, prolonged interference with drainage and the presence of cholesteatoma. Thus in the vast majority of instances we find either polypi, cholesteatoma or small tympanic perforations chiefly in Shrapnell's membrane. Furthermore, in chronic suppurative cases, there usually occurs a sclerosis of the cortex which favors necrosis of the deeper lying bony structures. It is particularly in those instances in which cholesteatoma has eroded the bone and lies in direct contact with the dura that intracranial complications occur. The cholesteatoma not only seriously interferes with drainage, but also acts as an excellent medium for the growth of the invading microörganisms. In acute middle-ear suppuration the mucous membrane and the bony walls are intact, and intracranial extension occurs only when the infection is so viru-

lent that thrombi are formed in the vessels which pass between the middle ear and cranial cavity. Occasionally, in acute middle-ear suppuration, the infection travels through the labyrinthine windows to the internal ear and then through the internal auditory canal into the cranial cavity. In such cases, however, meningitis is more apt to result than cerebellar abscess.

According to most authors cerebellar abscess is not as common as abscess in the temporo-sphenoidal lobe. Koerner found 69 cases of temporo-sphenoidal lobe abscess to 30 of cerebellar abscess. Growers found 186 temporo-sphenoidal lobe abscesses to 41 cerebellar. Le Fort and Lehmann found 327 temporo-sphenoidal lobe abscesses to 113 cerebellar. Michaelsen found cerebellar abscess to be as frequent as temporo-sphenoidal lobe abscess. Eight of his cases were in the cerebellum and eight in the cerebrum.

Males are attacked much more frequently than females, the proportion, in our series, being 3:1.

Author.	*0-10 yrs.*	*11-20 yrs.*	*21-30 yrs.*	*Over 30 yrs.*
Koch	11	33	35	19
Okada	17	56	45	28
Koerner	4	14	12	10
Heimann	16	57	38	26
Neumann	12	51	41	30

Neumann gives the above statistics on the relative frequency of occurrence of cerebellar abscess at different periods of life.

In the series we have collected, it occurs with greatest frequency in the second decennium, and with least frequency in the first and sixth decennia, while in the third, it occurs about as frequently as it does in the fourth and fifth combined. Thus in the 86 cases, 6 were under 10, 29 were between 10 and 20, 22 were between 20 and 30, 12 were between 30 and 40, 10 were between 40 and 50, and 5 between 50 and 60. In 2 the age was not stated. It occurred 45 times on the right side and 37 times on the left. In 4 the side was not stated.

With regard to the etiology of cerebellar abscess, Neumann differentiates between those which complicate the acute middle ear infections and those occurring with the chronic otitides. Of 19 cases of cerebellar abscess following acute middle-ear suppuration, the description was so imperfect in 4 that the pathway of infection could not be determined. Of the remaining 15 cases, 8 were due to sinus thrombosis, and 6 followed epidural abscess in the posterior fossa. In one there was a labyrinthitis, but it could not be positively determined that this was the cause of the abscess. Our statistics confirm this viewpoint. In the 14 cases of cerebellar abscess complicating acute otitis media and mastoiditis, sinus thrombosis occurs in 6. In only

one, however, is there a direct connection evident, macroscopically, between the infected sinus and the cerebellar abscess. This proportion is probably too low and, in other statistics, such evidence is more frequently met with. Although in the majority of instances the cerebellar infection is secondary to the sinus thrombosis, this does not seem to be invariably so.

Death from the rupture of a cerebellar abscess into the lateral sinus has been reported, and the fact that symptoms of sinus thrombosis occur late in some of the cases, notably those of Barr, Beck and others, brings to mind the possibility of the sinus infection being secondary to the brain abscess. Histological proof of this etiological relationship is not at hand. In four of the acute cases an epidural abscess in the posterior fossa is probably the pathway of the endocranial complication. So that in our series, too, the route, which the cerebellar infection follows, in the majority of cases complicating acute otitis, is either from a thrombosed sinus or an epidural abscess. In but one of the acute cases (Uffenorde) was the labyrinth the pathway of the intracranial infection. This was an acute middle-ear infection complicating scarlet, and the cerebellar abscess resulted from an extension of a saccus empyema. Here, despite the virulence of the infection and the lack of resistance on the part of the individual attacked, the intracranial extension

from the suppurative labyrinthitis was a circumscribed collection of pus, i. e., a brain abscess and not a diffuse purulent meningitis.

The cerebellar abscesses which complicate the chronic middle-ear suppurations differ very decidedly in their etiology from those which occur with the acute otitides. In Neumann's series, there are 132 cases of cerebellar abscess complicating chronic suppurative otitis media. Twenty are so incompletely described that they are worthless. Of the remaining 112, 49, that is 43.75 per cent, showed labyrinthine suppuration. Thirty-eight were due to sinus thrombosis and 16 to extradural abscess of the posterior fossa. In 3 cases there were extradural abscess and sinus thrombosis and in 6 cases there were labyrinthitis and sinus thrombosis. In the series collected by us, 31 of the 72 cases with chronic purulent otitis showed labyrinthine suppuration. These figures, startling as they are, are probably an underestimate rather than an overestimate. We base this judgment upon the following facts. If we examine individual reports included in our series, we find first 8 cases reported by Michaelsen. Of these, 6 are cerebellar abscesses complicating chronic suppurative otitis media. In these 6 cases "dead labyrinth" was found 5 times. Again Ruttin reports 7 cases, all of them complicating chronic suppurative otitis. In six of the seven, labyrinthine suppuration occurred. Neumann reports 5

cases, all of them complicating chronic purulent otitis media. In these, labyrinthine suppuration occurred three times. These figures, we believe, are the result of careful clinical examination and are not accidental. In many of the cases the labyrinthine suppuration was proven histologically.

Undoubtedly, there is a small proportion of cases of cerebellar abscess in which, although the labyrinth is the seat of a suppurative process, it is not the pathway of the infection from the middle ear to the endocranial structures. In a case which we recently had an opportunity to examine post mortem and in which the temporal bone was examined histologically, the following facts presented. Not a vestige of labyrinthine endorgans, auditory or static, remained. There was a fistula in the external semi-circular canal filled with granulations and new-formed connective tissue. From this focus the labyrinth probably became infected. The crura of the stapes were destroyed, but the foot-plate, although much eroded, was still present in the oval window. With the exception of this fistula in the external semi-circular canal, the bony canals were everywhere intact. There was, furthermore, no erosion of the inner labyrinthine wall. The internal auditory canal contained no pus. The pathway of the intracranial infection could be traced through the inner (posterior) antral wall. In this situation there was an epidural abscess and a

perforation led through the bony wall and through the dura to the cerebellar abscess.

In our series, suppurative labyrinthitis and epidural abscess occurred seven times. Thus suppurative labyrinthitis, epidural abscess and cerebellar abscess form a fairly common triad. The etiological relationship between these three factors is not a constant one. While it is natural to suppose that the epidural abscess is secondary to the suppurative labyrinthitis, this is not invariably so. In his consideration of the relationship between deep epidural abscess in the posterior fossa and suppurative labyrinthitis, Grünberg reports a most carefully studied case. The labyrinth was the seat of a diffuse suppurative process. The windows, both the oval and the round, were intact. The labyrinthine capsule was everywhere intact with the exception of the common limb, i.e., the junction of the superior with the posterior semi-circular canal. At this point there was an erosion from without inward, unquestionably caused by the epidural abscess in this location.

Ruttin has shown that in mastoiditis, particularly in those latent varieties due to infection by the streptococcus mucosus, there is a tendency to necrosis in the deep seated petrosal cells surrounding the labyrinthine capsule. Drainage from this region is particularly difficult and an epidural abscess may result. It can readily be understood, then, how an epidural ab-

scess in this location may give rise to cerebellar abscess on the one hand, and to a diffuse labyrinthine suppuration on the other.

Formerly, in many of these cases of suppurative labyrinthitis, epidural abscess and cerebellar abscess, a saccus empyema was adjudged, upon macroscopical findings, the pathway of the cerebellar infection. Macroscopical findings, however, are inadequate to establish such a judgment. The microscope alone is the final arbiter. In order to diagnose saccus empyema it is necessary to demonstrate an epithelial lining in the abscess cavity. In those cases in which the cerebellar abscess is secondary to a saccus empyema, there is a likelihood that a sinus thrombosis is also present. The pus in the saccus gradually extends backward between the two layers of the dura, this being the direction in which the fibrous tissue connecting the layers of the dura is least dense. The pus eventually comes to lie in the inner wall of the lateral sinus. In this situation it is located between the sinus and the cerebellum. The infection may extend in both directions causing a sinus thrombosis and a cerebellar abscess. When the clinical examination shows symptoms of suppurative labyrinthitis, sinus thrombosis and cerebellar abscess, the pathway of infection is usually through the saccus. Kramm and Hegener have reported such cases with histological examination.

In eight cases, suppurative labyrinthitis occurred with sinus thrombosis. In only one of these, however, is the labyrinth clearly indicated as the direct pathway of the sinus infection. In this instance, in addition to the sigmoid sinus, the bulb, inferior petrosal and cavernous sinuses were also thrombosed. Here the infection, in all probability, started in the labyrinthine veins, extended to the inferior petrosal sinus and thence forward to the cavernous and backward to the bulb and sigmoid. The pathways of infection from the middle ear to the cerebellum are as follows:

1. From the middle ear through the round or oval window or through a fistula in the horizontal semicircular canal or promontory to the internal ear. From here through a fistula in the posterior petrosal surface to the cerebellum.

2. From the middle ear to the labyrinth; through the internal auditory canal to the cerebellum.

3. From the middle ear to the labyrinth; through the ductus endolymphaticus to the saccus. From here to the cerebellum.

4. From the middle ear to the labyrinth; through the aqueductus cochleae to the cerebellum.

5. From the middle ear to the antrum and mastoid cells; through the inner (posterior) antral wall to the lateral sinus. From the sinus to the cerebellum.

6. From the middle ear to the antrum and mastoid cells; through the inner table to the epidural space

either in front of or behind the sinus. From here to the cerebellum.

7. From the middle-ear to the facial canal. From here to the cerebellum.

From an etiological point of view as well as from a pathological and clinical point cerebellar abscess may be divided into

a. Labyrinthogenic.

b. Non-labyrinthogenic.

In all probability at least 50 per cent of cerebellar abscesses complicating chronic supprative otitis media are labyrinthogenic. In a small proportion of cases, the labyrinth is the seat of diffuse suppuration and is destroyed, but is not the pathway of infection to the brain.

On the other hand, of those cases of cerebellar abscess which complicate the acute otitides, from 90 to 95 per cent are non-labyrinthogenic, i. e., they take origin either in a sinus thrombosis or an epidural abscess of the posterior fossa. In a general way, middle-ear infections which progress to the intracranial structures through natural openings, lead to a diffuse suppurative process, i. e., meningitis, whereas those that do not follow preformed paths cause circumscribed collections of pus, i. e., either brain or subdural abscesses. The aqueductus vestibuli offers a notable exception to this rule. An infection traveling along this route is retarded, probably because of the narrow

lumen of the aqueduct, the facility with which protective adhesions are formed and the fact that the aqueduct ends in a closed sac between the layers of a very resistent membrane, viz., the dura. Such infections, then, frequently lead to cerebellar abscesses, rarely through a saccus empyema, more often through an erosion of the bony wall of the aqueduct and the formation of an epidural abscess.

Whether the labyrinth infection leads to a diffuse meningitis or to a cerebellar abscess depends upon the virulence of the infection. In this regard the progress of the middle-ear infection into the labyrinth is of considerable importance. Slow, erosive processes, destroying the labyrinthine capsule, are much more apt to arouse circumscribed intracranial complications than are labyrinthitides in which infection takes place suddenly through the windows.

A notable example is the case reported by Thompson. In this instance, cholesteatoma had eroded both labyrinths, and the latter were the seat of diffuse suppuration. Each lateral cerebellar lobe contained an abscess. The pathway of the intracranial infection was along the nerves in the internal auditory canal. That the pathway of the infection into the labyrinth may be of importance in determining the character of the intracranial complication even where the latter occurs with the acute otitides, the case reported by Uffenorde illustrates. Here the labyrinth became

infected by an erosion through the promontory and not by the windows.

Of the labyrinthogenic cases of cerebellar abscess, those with a fistula in the posterior petrosal wall are by far the most common. The suppurative process does not extend, in these cases, from within one of the semi-circular canals or the vestibule, through the posterior petrosal wall by a process of erosion; but the sequence, according to a number of cases examined histologically by Hegener, is as follows: As a result of the chronic inflammatory process in the middle-ear (usually with cholesteatoma), there is an erosion of the labyrinthine capsule with the formation of a fistula, most commonly in the horizontal semi-circular canal. The intralabyrinthine space becomes filled with pus and granulations, the membranous labyrinth being destroyed. Following extension of the granulation tissue into the fundus of the internal auditory canal, the internal auditory artery becomes occluded. As this artery constitutes the entire blood supply of the internal ear, there results a necrosis of the bony labyrinthine capsule. This dead bone sets up a reactive inflammation in the surrounding spongy bone and very soon the necrotic labyrinthine capsule becomes enveloped in a zone of granulation tissue. This reactive inflammation in the spongy bone surrounding the labyrinthine capsule gradually extends to the posterior petrosal surface and finally reaches the dura.

Cerebellar abscess is usually situated near the ear which is the origin of the infection. The only exceptions to this rule are those cases in which the abscesses are pyemic in origin. According to Koerner's statistics, in 42 per cent of the cases of brain abscess observed by him, there was a fistula between the brain abscess and the suppurating focus in the temporal bone. In 15 per cent of the cases the brain substance between the abscess and the dura was broken down so that only the dura remained between the abscess and the diseased bone. In 15 per cent the brain was adherent to the membranes in the affected region and in 17 per cent the brain substance between the abscess and the dura was softened and discolored.

Abscesses are usually situated in the anterior portion of the cerebellum. Those cases which are secondary to sinus thrombosis or to epidural abscess are usually located in the lateral hemisphere. Those which are secondary to labyrinthitis usually involve the vermis or the mesial portion of the lateral hemisphere.

One of the most interesting problems connected with the pathology of brain abscess in general, but particularly with cerebellar abscess is that concerning the process by which the brain substance itself becomes infected.

Cerebellar abscesses may be divided into two classes viz.: superficial and deep. The former are situated in

the cortex of the cerebellum and the latter in the central white substance. The pathogenesis of these two forms is entirely different.

The superficial abscesses are formed as follows: the purulent process in the temporal bone passes into the posterior fossa, forming an epidural abscess. There follows an extension of the inflammatory process through the dura with the formation of adhesions between the dura, pia-arachnoid and surface of the cerebellum. The next step is a perforation through the dura and the formation of an abscess between the dura and pia-arachnoid. This is called a subdural abscess. The pia-arachnoid and the superficial layers of the cerebellar cortex then break down and suppurate and thus a superficial cerebellar abscess results. This type of abscess is sometimes called a meningo-encephalitis. It is far less common than the second class or deep cerebellar abscess. All who have observed a number of cases of brain abscess have been impressed with the fact that in most instances the collection of pus is more or less remote from the cortex, i. e., that the majority of brain abscesses are situated in the white substance. To those who have performed post-mortem examinations on a number of these cases it has become evident that, between the brain abscess and the source of the intracranial infection, there is frequently a layer of brain substance which is macroscopically normal. In other words there is a growing

impression that the majority of brain abscesses do not occur through direct extension by continuity.

Even where there is a post-petrous epidural abscess and where the brain surface is grown fast to the dura there is often, interposed between the epidural abscess and the brain abscess, a layer of normal brain tissue. Thus Isemer in his case report states: It is remarkable that, although the brain surface is grown fast to the dura in the region of the epidural abscess, yet between the white substance and the area of the dura there is a distinct, if thin, layer of macroscopically normal brain. From this one might conclude that the brain abscess was not the result of an extension by continuity from the diseased labyrinth, but that the infection extended by thrombosis of small cerebellar veins which empty into the pial veins, or through the lymph sheaths around these veins.

With regard to this question Michaelsen (quoting Uffenorde) states: The origin of the brain infection is always the meninges, i. e., the dura and the pia-arachnoid. At first, the pial veins become involved and thrombosed. This thrombosis which, in the beginning, is not always septic, follows the venous channels backwards, leaving the cortex intact, until it reaches the ultimate ramifications of the veins in the white substance, where the stasis, producing red softening, results in an extravasation of red and white blood cells. Bacteria then pass into this area, the brain tis-

sue breaks down and an abscess results. The cortex is not involved because of its separate vascular system. Instead of the veins being the carriers of the infection, the latter may travel along the perivascular spaces or the lymphatic vessels. Sometimes a thrombus forms in an artery and a piece of this is carried off and lodges in a terminal vessel in some part of the cerebellum. Here it causes necrosis of the brain tissue, as the cerebral vessels are terminal vessels. The addition of bacteria in the necrotic area results in the formation of an abscess. A case of this kind in which the abscess was located in the frontal lobe of the brain was seen by us through the courtesy of Dr. Berens and reported by him in the *Annals of Otology, Rhinology and Laryngology,* 1914. As a result of a chronic middle-ear suppuration there occurred an erosion of the internal carotid artery where it lies in close relationship with the inner wall of the bony Eustachian tube. An infected thrombus formed in the artery. A portion of this was washed away by the blood stream and lodged in a terminal vessel in the frontal lobe of the brain. Here it caused an area of necrosis with the formation of a large abscess.

Even though the "origin of the brain infection is always the meninges," yet the appearance of the dura and its behavior during the post-operative treatment often give absolutely no indication as to the presence of a brain abscess. Collections of pus within the brain,

with a macroscopically normal dura are not so rare. In a case of right temporo-sphenoidal abscess recently observed by us, the dura was disclosed at operation covered with cholesteatoma and large pussy granulations. During the post-operative dressings the dura returned to normal, i. e., it became coated with small red healthy granulations and healing was uneventful. Two months after the radical mastoid operation the brain abscess ruptured into the lateral ventricle and death ensued from diffuse purulent meningitis.

The observations of Heilbronn, although too isolated to be conclusive in themselves, throw a flood of light upon the modus operandi of brain infection through the blood vessels.

He states: "There are few observations of brain lesions which have occurred through venous channels (Rücklaüfigem Wege). Such affections we would expect chiefly in disease of the great sinus, the chief return current of the brain's venous blood. With the most frequent form of sinus disease, i. e., thrombosis and thrombo-phlebitis, there have been few observations of brain disease. Oppenheim in his 'Lehrbuch der Nervenkrankheiten' states that he believes there are many opportunities in cases of sinus thrombosis to observe focal brain symptoms. Hemorrhage and softening are often the direct result of sinus thrombosis, and, although such observations are rare, they occur far more frequently than they are observed."

Heilbronn reports several interesting cases with general and focal brain symptoms complicating otitic sinus thrombosis. In the first case, a patient with acute suppurative otitis media and mastoiditis, symptoms of meningitis followed six days after a simple mastoid operation had been performed. With a gradual rise of temperature, the patient became delirious and finally comatose. Post-mortem examination showed no meningitis. There was a thrombosis of the left sigmoid sinus. There were hemorrhages in both optic thalami, thrombosis of the venæ Galeni and of other basal veins. The symptoms were due to the disturbance in the intracerebral circulation. He draws the following conclusions:

1. In sinus thrombosis by the plugging of the small veins, lesions of the brain in the form of hemorrhages and softening may occur.

2. These lesions, like many other brain lesions, may cause general brain symptoms without definite focal symptoms.

The second case was that of a young man in whom a sinus thrombosis complicated a chronic suppurative otitis media. He suddenly became unconscious. This condition, however, lasted but a few hours and was followed by an aphasia. Post-mortem examination showed extensive sinus thrombosis. The large pial vein which surrounds the left temporo-sphenoidal lobe and has its origin in this lobe was thrombosed.

The aphasia was due to the occlusion of this vein and stasis of the blood with the consequent nutritional disturbance. The third case, probably the most significant, was that of a young woman in whom sinus thrombosis complicated a chronic suppurating ear. In this instance there were signs pointing to a bulbar lesion. Anatomically there was no gross obstruction of the veins leading from this region, but in the medulla there were changes which doubtless indicated a destruction of function of this part. There were destruction of fibers and a paucity of nuclei, while the remaining nuclei stained poorly and were deformed. The picture was one of anemic necrosis. The lesions were significantly distributed about the vessels. To the center of each focus ran a small vessel.

These observations, while they are, as stated, too isolated to be conclusive, yet offer strong confirmation of Uffenorde's theory. Whether this theory, regarding the manner in which the brain tissue itself is infected, holds good only for those cerebellar abscesses which take their origin in a thrombosed sinus, or whether it can be applied to all cases irrespective of their origin, remains to be proven. Only careful histological research will clear this point.

Cerebellar abscesses are usually irregular in shape. They often have pockets or diverticula, which may be connected with the main abscess by means of a narrow stalk. Such a diverticulum may give the impression

of a secondary abscess, but multiple abscesses in the cerebellum are unusual except when they are pyemic in their origin. Koerner found multiple abscesses in four cases out of thirty-two cerebellar abscesses.

The brain tissue surrounding the abscess is actively inflamed. The vessels are hyperemic and some of them are thrombosed. Around the vessels are masses of extravasated red and white blood cells. There is some edema. This encephalitis causes an increase in the size of the affected hemisphere and pressure upon the remaining structures in the posterior fossa. The vermis, pons and medulla are often pushed to one side of the median line. Pressure of the enlarged cerebellar hemisphere upon the veins in the posterior fossa may cause sufficient interference with the return circulation from the pia lining the ventricles to cause an internal hydrocephalus.

At the periphery of the abscess the brain tissue is necrotic and broken down. The surface of the brain tissue directed toward the abscess cavity is ragged. Thrombosed vessels project into the cavity. The thrombosis in the vessels at the periphery of the abscess prevents bleeding into the cavity. When the breaking down of tissue is more rapid than the thrombus formation hemorrhage occurs.

When the breaking down of the tissue has ceased and the brain tissue has sufficient vitality to manifest

a tendency to reparative processes, a network of fibrin is formed at the periphery of the abscess. This fibrin network is formed from the elements of the surrounding living tissue. Leucocytes pass into this network, gradually become spindle-shaped and finally change to connective-tissue cells. In this way a capsule is formed about the abscess. At first the capsule is non-vascular. Later, primitive blood vessels pass into the capsule from the periphery. The membrane gradually becomes thicker and separates the abscess from the normal brain tissue. The growth of the abscess is usually slower after the capsule has formed, and may cease altogether. The capsule may be from 1 to 5 mm. or more in thickness. The older the abscess, the thicker the capsule, as a rule. A capsule has been found in abscesses which were only five or six weeks old. Sometimes the periphery of the capsule becomes very dense and fibrous with very few blood vessels, or it may even become calcified.

The histological appearance of a typical abscess capsule is described by Oppenheim as follows: "Three zones are seen in the capsule. The inner granulation zone shows no sharp demarkation from the abscess cavity. It consists of numerous round cells with very little intercellular substance and very few vessels. The second zone consists partly of wavy and partly of straight fibers. In the central portion of this zone the fibers are closely packed and in the peripheral por-

tion, more loosely arranged. Between the fibers, in addition to spindle cells, there are cells which represent partially changed glia cells and round cells. In this layer there are numerous thick-walled blood vessels. The fibroblasts which form the fibrous tissue, seem to belong only to the mesodermal tissue (vessel walls and connective tissue surrounding the vessel walls), whereas the neuroglia seems to take no part in this formation. The third or outer zone consists of a more uniform ground substance which contains numerous round cells and large swollen glia cells. In no place is there a sharp demarkation between the healthy tissue and the capsule. Everywhere there are signs of an active process and nowhere is there real cicatrization."

Not all brain abscesses have capsules. The presence of a capsule does not prevent an increase in the size of the abscess. The capsule may be replaced by exuberant granulations, pus may be formed anew, and thus the abscess may enlarge. The capsule stretches and may finally rupture, the abscess emptying itself into the fourth ventricle or the subarachnoid space.

An encapsulated abscess sometimes acts as a foreign body and sets up irritative changes in the surrounding tissue. Thus a second abscess may be formed in which the first encapsulated abscess is found floating. Encapsulated abscesses may remain

latent for a long time, sometimes several years, and then enlarge and rupture.

When a fistula from the mastoid communicates with the interior of the cerebellar abscess, it is not the result of a breaking down of tissue from the surface to the interior of the abscess. The abscess ruptures onto the surface of the cerebellum, forming first a subdural abscess, and then perforating through the dura into the mastoid cavity.

Around the abscess, the brain tissue is always more or less inflamed. This encephalitic area may be very extensive. The encephalitis may extend and cause death even after the abscess has been evacuated. It manifests itself macroscopically, on cross-section of the brain, by swelling of the brain tissue and by fine red dots resembling flea-bites, scattered through the white matter. In a histological examination of such an encephalitic area, Dupré and Deveaux found diffuse degeneration of the cortical cells without involvement of the nerve fibers. They concluded that these changes are due to toxins which arise from the abscess.

The contents of the abscess consist of pus cells, many of which are undergoing fatty changes, large mononuclear lymphocytes, red blood cells and free nuclei. Between the cells is an amorphous exudate, fragments of dead tissue, hematoidin, margarin and cholesterin crystals and bacteria.

Many kinds of bacteria have been found in cerebellar abscess. Streptococcus pyogenes and staphylococcus pyogenes aureus are the most common. Staphylococcus pyogenes albus and citreus are less common. Streptococcus mucosus capsulatus, pneumococcus and bacillus of Friedlaender occur. The same organisms may be found in the abscess as occur in the middle-ear suppurations. Bacillus pyocyaneus, streptococcus pyogenes fetidus, micrococcus pyogenes tenuis, bacillus typhosus, bacterium coli and bacillus proteus vulgaris have been found.

Mixed infections often occur. Saprophytic organisms are sometimes found together with pyogenic organisms. Occasionally saprophytic organisms are found alone. When this occurs, it is probable that pyogenic organisms were present, but have died out. Occasionally no bacteria are found in the abscess. Tubercle bacilli have been found.

According to Neumann, when the abscess is due to diplococci, fibrinoplastic changes occur, which result in well-marked demarkation and encapsulation. When it is due to anaërobic bacteria, there are soft necrotic margins and no attempt at encapsulation. The anaërobes produce sulphuretted hydrogen, which gives the fetid odor to some brain abscesses. These anaërobic bacteria flourish in the mouth as harmless saprophytes and pass into the middle-ear through the Eustachian tube.

In some cases, different organisms were found in the middle-ear, brain abscess and spinal fluid. In the same abscess different bacteria were found at different times.

In acute abscesses, the cavity of the abscess becomes obliterated as soon as it is evacuated, as a result of the pressure of the surrounding swollen brain tissue. In chronic abscesses, with rigid walls, it may take many weeks before the abscess cavity is obliterated.

The cases of brain abscess which evacuate themselves spontaneously, through a fistula into the mastoid, and thence through a perforation in the drum-membrane, or through a fistula in the skin over the mastoid, do not heal, as the fistula is so narrow and tortuous that it does not provide adequate drainage.

Very rarely, according to Macewen, a brain abscess may heal spontaneously and become absorbed. Blood vessels from the normal brain tissue may penetrate the abscess capsule and cause resorption of the pus through phagocytosis.

CHAPTER IV

SYMPTOMS OF CEREBELLAR ABSCESS

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THE symptoms of cerebellar abscess divide themselves into two groups. In the first group must be placed those symptoms which are common to all forms of intracranial complications. The second group consists of those phenomena which are peculiar to cerebellar lesions and are the result of disturbance in cerebellar function. Lesions of the fiber tracts in the cerebellum which connect the vestibular nuclei with the cerebellar nuclei and the cerebellar cortex give rise to symptoms similar to those caused by lesions of the vestibular centers or of the static labyrinth. Consequently the focal symptoms of cerebellar abscess may be divided into two groups.

A. Symptoms of disturbances in movements.

B. Symptoms of disturbances in the vestibular apparatus.

A. The symptoms of disturbance of movement are the following:

1. Hypermetria.
2. Asynergy.
3. Adiadokokinesis.

4. Tremor.
5. Disturbances in writing.
6. Disturbances in speech.
7. Atony or hypotony.
8. Catalepsy.
9. Spontaneous deviations of the extremities and loss of reaction movements.
10. Spontaneous falling and loss of reaction movements of the trunk.
11. Hemiparesis.
12. Fixed attitude of the head.
13. Disturbances in weight estimation.

B. The symptoms which are due to destruction of the fiber tracts between the vestibular nuclei and the cerebellar nuclei are:

1. Nystagmus.
2. Enduring nystagmus.
3. Vertigo.
4. Vomiting.

The general symptoms of cerebellar abscess may be divided into two groups.

A. Those due to increased intracranial pressure.

B. Those due to the inflammatory process in the cerebellum.

A. Symptoms due to increase in intracranial pressure are:

1. Headache.
2. Vomiting.

3. Disturbances of the sensorium.
4. Slow pulse.
5. Disturbances of respiration.
6. Optic nerve changes.
7. Paralysis of the cranial nerves.
8. Changes in the reflexes.

B. The symptoms due to the inflammatory process in the cerebellum are:

1. Changes in temperature.
2. Emaciation.
3. Changes in the blood.
4. Changes in the cerebrospinal fluid.

Headache is the most constant of the symptoms of cerebellar abscess. It was mentioned in 70 cases out of the 86 collected. It is usually intermittent as are most of the general symptoms. Occasionally the headache is dull and boring with exacerbations which are unbearably severe. There may be periods during which the patient is entirely free from pain. The location of the headache is not characteristic but it is most commonly occipital. Sometimes there is tenderness to percussion of the skull over the site of the abscess.

Vomiting is more persistent with cerebellar abscess than with abscess in the temporo-sphenoidal lobe. It was mentioned in 48 out of the 86 cases. It may occur with or without nausea. There are two types of vomiting with cerebellar abscess, viz., projec-

tile, which is due to the increased intracranial pressure and vomiting accompanied by nausea, which is due to involvement of the vestibulo-cerebellar tracts. The vomiting bears no relationship to the ingestion of food. Nausea without vomiting was mentioned in four cases.

Mental dulness and somnolence are rarer with cerebellar abscess than with cerebral abscess. In all probability the mental changes are toxic in origin and are not due to pressure. Drowsiness was noted in 32 of the cases. Delirium or hyperexcitability is rare.

Bradycardia is a symptom frequently met with in cerebellar abscess. It was mentioned in 38 of the cases. The pulse is not uniformly slow during the entire 24 hours. While during most of the day, it may range between 60 and 75, for a short time it may drop to 50 or even lower. This drop often occurs during the early morning hours. The pulse is always slow in relation to the temperature, so that even with a complicating meningitis and a temperature of 103° F. or 104° F., the pulse is not likely to be above 70.

Pressure of the abscess on the medulla sometimes causes disturbances of respiration. Respiration may be as low as 10 or 12 to the minute. It may take on the Cheyne-Stokes type. In a number of cases respiration suddenly ceased during operation for cerebellar abscess while the heart continued to beat.

Optic nerve changes were present in 16 of the cases. The condition found is usually an optic neuritis but occasionally there is choked disc. It may be unilateral or bilateral, and if unilateral, may be on the affected side or on the opposite side. The impairment of vision is usually not very great. Optic nerve changes occur a great deal more frequently in cerebellar abscess than in abscess of the temporo-sphenoidal lobe.

Paralyses of the third, fifth, or sixth nerves were present eleven times. Inequality of the pupils was present in four of the cases, and in two the pupils were dilated and unequal. Anesthesia of the cornea occasionally occurs. These paralyses are due either to an accompanying basilar meningitis or to pressure upon the nerves. In some cases there is present, instead of a third or sixth nerve paralysis on one side, a conjugate deviation of the eyes. This may be accompanied by a deviation of the head. Facial paralysis or paresis occurred in 27 cases. In the majority of these, the paralysis was due to involvement of the facial nerve in the temporal bone, as the result of the accompanying labyrinthine suppuration. In a few cases it was due to pressure on the intracranial portion of the nerve. The difficulty in swallowing, which occurs occasionally, may be due either to pressure on the glossopharyngeal nerve or to pressure on the medulla. Changes in the reflexes

were mentioned in 7 cases out of the 86. They were increased in 5 and diminished in 2.

The temperature was noted in 55 of the cases. It was above 100° F. in 23. It was between 98.6° F. and 100° F. in 21 cases. It was subnormal in 11 cases. The higher temperatures usually occur in the terminal stages after a meningitis has set in. A persistently normal temperature does not exclude cerebellar abscess.

Partly, perhaps, because of the persistent vomiting, but much more because of the profound trophic disturbance, cerebellar abscess causes a rapid and extreme emaciation. The patients are cachectic in appearance. They are usually constipated, with coated tongue and fetid breath. There is rarely any bladder disturbance except just before death.

The blood changes with cerebellar abscess are not characteristic. There may be a leucocytosis with a high polynuclear count, or the blood picture may be normal. No cases of bacteriemia have been reported in uncomplicated cases of cerebellar abscess.

The cerebrospinal fluid was examined in 24 of the cases. It was normal in 9 of the cases; it was normal but under increased pressure in 2 cases; it was cloudy and sterile in 8 cases. In 3 of these the fluid was under increased pressure. It contained bacteria in 5 cases. In our own experience a cloudy, sterile fluid

was the most common finding in uncomplicated brain abscess.

The focal symptoms of cerebellar abscess are due to,

1. Destruction of brain tissue.
2. The encephalitis surrounding the abscess.
3. Pressure of the abscess on the surrounding tissues.

The symptoms due to actual destruction of brain tissue are permanent. Those due to encephalitis and pressure may disappear after the abscess is evacuated.

The symptoms of disturbance of movement are the following:

1. *Hypermetria.*—The cerebellum seems to exercise an inhibitory influence on movements. In cerebellar lesions, there is a suppression of this inhibition, resulting in unmeasured or immoderate movements. This is called hypermetria. It may occur in spontaneous movements, but is much more likely to occur in certain commanded movements. For instance, if a patient with cerebellar abscess is asked to touch the tip of his nose with the end of his index finger he will point past the nose. A similar hypermetria due to overflexion of the thigh may be observed when a patient attempts to touch the knee with the opposite heel.

If a patient is seated, with the palm of his hand on the knee of the same side, and he is asked to supinate the hand so that the back of his hand rests on the same

spot where the palm was, the movement will be overdone, and the ulnar side of the hand will lie higher than the radial.

A vertical line is drawn near the right edge of a sheet of paper. The patient is asked to draw horizontal lines, starting anywhere, but ending at the vertical line. The patient will continue all of his lines past the vertical line.

In walking, at the beginning of the step, flexion of the thigh on the pelvis is more pronounced than normal, leading to excessive raising of the foot. At the end of the step, the sole of the foot makes a greater noise than normal in striking the floor, denoting excessive extension of the thigh.

The hypermetria is best brought out if the patient is asked to perform the movement quickly, for, if it is done slowly, he can correct the error.

Vision has no effect on the movements. The same error is made with the eyes open, as with the eyes closed.

The direction of the movement is conserved. It passes the mark, and then deviates.

2. *Cerebellar Asynergy.*—If a patient with cerebellar abscess is supported on either side, without his movements being influenced, and asked to walk, he will stop at his first step. The thigh is flexed and the foot is carried forward, but the upper part of the body does not move in harmony with the legs, the

trunk remaining extended on the thighs. Thus, having placed his foot on the ground with much noise, at the end of his first step, he can go no further. He is in danger of falling backward. It is necessary for the assistant to push the upper part of the body forward, or the patient can help himself by pulling his trunk forward with his arms, his hands catching some support in front of him.

If a patient, standing up, is asked to bend his head back and curve his body backward, he falls, because he only bends the trunk back, without bending the legs on the feet and the thighs on the legs.

If a patient with a cerebellar lesion lies on his back, with his arms crossed on his breast, he cannot sit up. He flexes the thighs at the hip, and lifts the heels from the floor.

These tests show that a patient with cerebellar abscess cannot synchronize the movements at the various joints in such a way as to perform an effective complex movement. Closing the eyes does not change this.

3. *Adiadokokinesis.*—This is the abolition or the diminution of the faculty of executing rapidly successive voluntary movements.

In a cerebellar lesion, muscular force is intact. The patient can execute as rapidly as a normal individual the elementary movements, pronation or supination. But the complete movement (pronation and supina-

tion) he performs more slowly than a normal individual. The phenomenon becomes more manifest if the act is repeated a number of times.

The symptom is present only in an individual who can perform the isolated movement, pronation or supination, with normal rapidity. For if he cannot do this, he cannot, of course, perform a rapid succession of the two movements.

It is sometimes bilateral, and sometimes unilateral. In the latter case, it occurs on the same side as the cerebellar lesion.

4. *Tremor.*—On attempting to point to an object with one of the extremities, orientation is correct, but the movement is not uniform. There are oscillations in different directions. The tremor occurs only during voluntary contraction of the muscles. It varies in different individuals, and in the same individual at different times. It may diminish, if the patient is left to himself, and not observed, and if he executes movements slowly and carefully.

On pointing to the nose with the finger, the movement to the nose may be direct, and then, at the end, there may be violent tremor.

5. *Disturbances in Writing.*—If one ask a patient with a cerebellar lesion to make a dot with a pencil, on a sheet of paper, he rarely succeeds at the first attempt. At first he either breaks the point or forces it through the paper. At the second attempt, he stops

above the paper. Finally, finding the surface of the paper, he makes, not a dot, but a line.

Cerebellar patients place commas and not dots over their i's. This is hypermetria. If they are asked to draw a line of a certain length, they will extend it further.

In attempting to draw a circle or an arc, as in the letters O or C, the figure is polygonal.

In attempting to reproduce regular zigzags, as in m and n, the angles are badly formed, and the lines irregular. Having arrived at the end of a line, the patient finds it difficult to begin a new line in the opposite direction.

The irregularities in writing are the result of tremor, hypermetria, asynergy and adiadokokinesis.

6. *Disturbances of Speech.*—Speech is scanning, jerky, a little explosive, and sometimes dragging. It is similar to that in disseminate sclerosis.

7. *Atony or Hypotony.*—Hypotony is present in a few cases. It is sometimes bilateral and sometimes unilateral, in which case it is on the side of the lesion. An experiment for determining the presence of hypotony of the extensors of the arm is as follows: Ask the patient to flex the forearm, and oppose this by traction on his forearm. Suddenly stop the traction. In a normal person, there results pronounced flexion, followed by a violent extension. In cerebellar lesions, this extension is only indicated.

8. *Cerebellar Catalepsy.*—This is a rare symptom. In certain positions where volitional equilibrium is realized, the voluntary muscles are held immobile for a long time without being contracted. It often appears as follows: The patient lies on his back, thighs flexed on pelvis, legs lightly flexed on thighs, feet separated. When he assumes this attitude, his legs and trunk oscillate in various directions, especially from left to right and vice versa, but at the end of several seconds, the body and lower limbs become fixed. This fixity is more marked than a normal individual can attain. It persists for several minutes, and causes no sensation of fatigue.

In cerebellar affections, volitional kinetic equilibrium is diminished, while volitional static equilibrium is preserved or even increased.

9. *Spontaneous Deviation of the Extremities, and Loss of Reaction Movements.*—These symptoms, as worked out by Barany, are of extreme importance in the diagnosis of cerebellar abscess. (Figs. 15-20.)

The method of examination is as follows: The patient is seated with his eyes closed. His arm is extended before him, and the index finger is made to touch the palmar surface of the examiner's index finger. The arm is then lowered until the hand touches the knee, and then raised until the finger again touches the examiner's finger, which, meanwhile, has not been moved. With a normal cerebellum, the

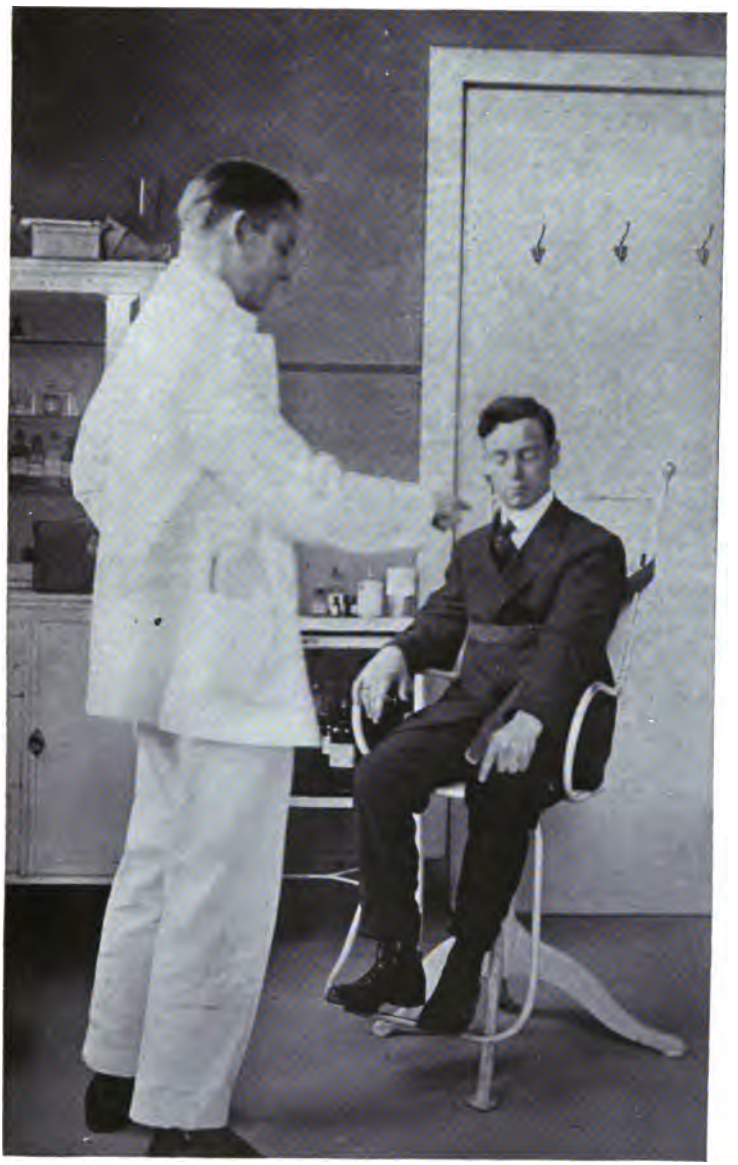

FIG. 15.—POINTING TEST FOR ROTATION.

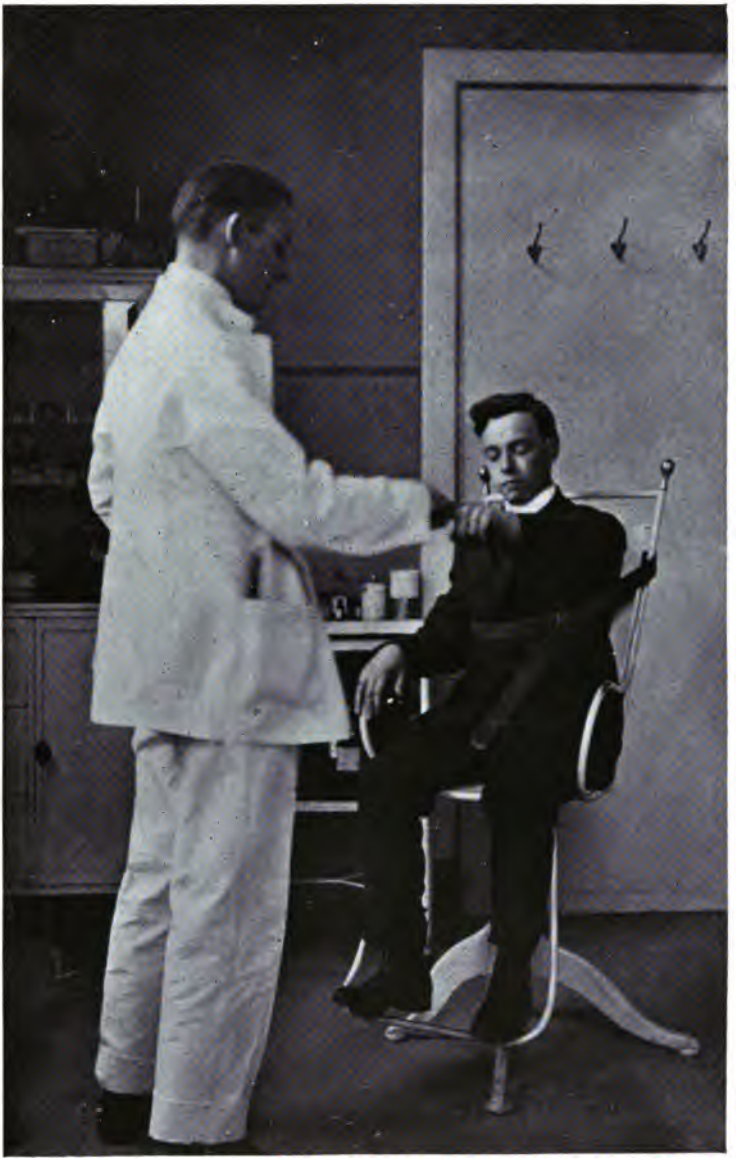

FIG. 16.—LOSS OF POINTING REACTION AFTER ROTATION.

FIG. 17.—NORMAL POINTING REACTION AFTER ROTATION TO LEFT.

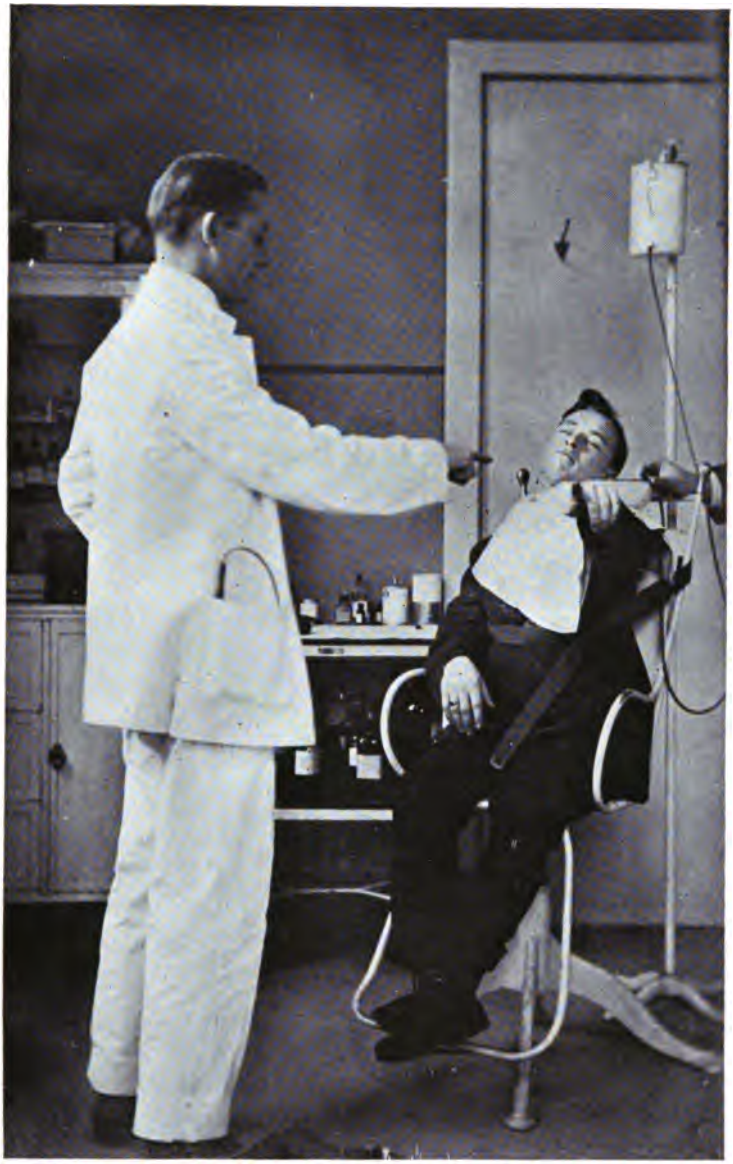

FIG. 18.—POINTING REACTION WITH CALORIC VESTIBULAR STIMULATION.

FIG. 19.—POINTING REACTION AT ELBOW JOINT.

FIG. 20.—POINTING REACTION AT WRIST JOINT.

patient points correctly. With a lesion in the shoulder area of the cerebellar cortex, the arm will deviate either inward or outward. If the lesion is in the right biventral lobe, the right arm will deviate outward, or to the right. If the lesion is at the middle of the margin of the right cerebellar hemisphere, i. e., at the middle of the right postero-superior and postero-inferior lobes, the right arm will deviate inward, or to the left. The former area contains the center for inward tonus of the shoulder joint and the latter the center for outward tonus of the shoulder joint. The left arm will point correctly. The deviation involves only the side of the body on which the lesion occurs. The movements of the elbow joint are examined by resting the elbow on the back of a chair, and moving the forearm. The movements of the wrist joint are examined by resting the wrist on the back of a chair, and moving the hand at the wrist. The wrist joint is examined with the hand in pronation, and then with the hand in supination, there being a separate center for each position of the hand. The wrist center lies in front of the arm center.

The hip is examined in a similar way. The hip center is behind the arm center. These tests should be made repeatedly in order to make sure that there really is a deviation, and the patient must not know when he has made an error in pointing as he will then make an effort to correct the error.

When the lesion in the cortex is a large one, many joints will be involved in the deviation. When the lesion is small, only one or two joints will be involved.

Having determined the presence of a spontaneous deviation, the next step is to test the reaction movements of the extremities. This is done by arousing a vestibular impulse.

If the patient is rotated to the left, he will have an after-nystagmus to the right. If the patient's arms are now held in front of him, they will both deviate to the left, i. e., in the direction of the slow component of the nystagmus, provided the cerebellum is normal. If, however, the patient has a lesion in the right cerebellar cortex involving the center for inward tonus of the shoulder joint, the right arm will point correctly, while the left arm will deviate to the left.

Therefore with destruction of this center, there is a spontaneous deviation of the arm outward, and a loss of the reaction movement inward.

Instead of rotation, the caloric test may be used to arouse the reaction movements, the head being tilted back 60°, in order to elicit a horizontal nystagmus. If a cold caloric is done in the left ear, and the head held upright, the arms held in front of the body will deviate to the left. If the right arm is moved laterally, it will deviate upward. If the left arm be moved laterally, it will deviate downward.

If the head is now rotated to the right shoulder,

both arms held in front of the body will deviate downward. If the head is rotated to the left shoulder, both arms held in front of the body will deviate upward.

The deviation depends upon the plane and direction of the nystagmus, and the position of the head. It is always in the plane of the nystagmus, and in the direction of the slow component of the latter.

10. *Spontaneous Falling, and Reaction Movements of the Trunk.*—With an abscess involving the worm, there is a tendency to fall. The falling may be in any direction. The direction of falling was noted in 13 out of the 86 cases collected. Of these, 10 cases fell to the side of the lesion, and 3 cases to the opposite side. If spontaneous nystagmus is present, the direction of the falling does not bear any definite relation to this nystagmus.

If an artificial rotatory nystagmus is aroused by means of cold or hot water in the ear, the patient falls in the direction of the slow component of the nystagmus, when the vermis is intact. For instance, if cold water is allowed to flow into the right ear, the patient falls to the right. If the head is turned to the left shoulder, the patient falls forward. However, if there is a lesion of the vermis, and the patient falls, for example, backward, the direction of the falling would not be changed by the production of a caloric vestibular impulse. In other words, the falling reaction is wanting. There may be an absence of the falling

reaction without spontaneous falling. The loss of the falling reaction may be in one or several directions. In the latter case, the lesion would be larger and more centrally located than in the former.

In order to differentiate between focal lesions and symptoms due to pressure from a distance, we must make a careful examination of the spontaneous symptoms as well as a careful functional examination. If there is spontaneous falling or deviation of the extremities, and on arousing the proper vestibular impulse, a falling or deviation in the opposite direction is elicited, we are dealing with a distant lesion. If, on examination at different times, there is sometimes deviation and falling and sometimes none, we are surely dealing with a distant lesion. If only a portion of the symptoms change, there is a combination of focal lesion and lesion at a distance. If, after a decompression operation, the symptoms disappear, there is a lesion at a distance.

Constant symptoms which increase in severity are due to focal lesions.

11. *Hemiparesis.*—This symptom occurs occasionally in cases of cerebellar abscess. It was noted in 6 cases out of the 86 collected from the literature. It is always on the same side as the cerebellar lesion.

12. *Fixed Attitude of the Head.*—In a few cases of cerebellar abscess, the head is held stiff and rigid, and

bent toward one shoulder. The cause of this attitude is not definitely known. It may be due to increased tonus of the neck muscles of one side. In experiments on dogs, where half of the cerebellum is removed, one of the most striking symptoms is the lateral deviation of the head and the lateral curvature of the entire body.

13. *Disturbances in Weight Estimation.*—A symptom due to disturbance of the sensory impulses was first described by Lotmar in the *Monatsschr. f. Psychologie u. Neurologie,* 1908, and later confirmed by O. Maas and others. This is the underestimation of weights by the hand on the side of the cerebellar lesion. A number of metal discs are prepared which are the same in size but differ in weight. A disc is put into each hand and the patient asked to judge whether they are equal or not. With a cerebellar abscess on the right side, a heavier weight in the right hand will seem equal to a lighter weight in the left hand. This symptom must be judged very carefully, for in right-handed individuals the left hand normally overestimates weight.

The symptoms which are due to destruction of the fiber-tracts between the vestibular nuclei and the central nuclei of the cerebellum are nystagmus, enduring nystagmus, vertigo and vomiting.

The *nystagmus* due to cerebellar disease cannot be distinguished from that due to disease of the vestibu-

lar apparatus. It varies in its direction. Nystagmus was noted in 46 out of the 86 cases. It was directed to the diseased side 15 times, to the sound side 7 times, to both sides 22 times, and in 2 cases the direction was not mentioned.

Of 40 cases of cerebellar abscess reported by Neumann, the nystagmus was directed toward the diseased side 17 times, in 14 cases the direction was not stated, and in 9 cases there was no nystagmus.

Although the appearance of cerebellar nystagmus does not differ from that of labyrinthine nystagmus, yet its behavior does. Labyrinthine nystagmus, when due to a diffuse labyrinthitis, gradually diminishes in intensity, and ceases in a few weeks. The direction of such a nystagmus is always away from the diseased ear. Cerebellar nystagmus remains the same or becomes more marked. A nystagmus toward an ear of which the labyrinthine functions are destroyed, cannot be labyrinthine in origin. It must be retrolabyrinthine, probably cerebellar. In some cases, there is a dead labyrinth with nystagmus toward the sound side. During the course of the disease, the nystagmus suddenly changes to the diseased side. This usually indicates a cerebellar abscess. Sometimes with cerebellar abscess there is a nystagmus in both directions, rotatory to one side and horizontal to the other.

The nystagmus of a circumscribed labyrinthitis is difficult to differentiate from that of a cerebellar ab-

scess. In the latter case, we often have other symptoms of intracranial disease to guide us. Neumann recommends doing a labyrinthectomy in cases of doubt. If there was a circumscribed labyrinthitis, the nystagmus will shift toward the sound side. If the nystagmus remains directed to the diseased side, we are dealing with a cerebellar abscess.

Enduring Nystagmus.—If cold water be allowed to flow into an ear, and the flow stopped as soon as nystagmus sets in, the nystagmus will continue from one to two and one-half minutes under normal conditions. In cerebellar disease, the caloric nystagmus which is directed toward the affected hemisphere will continue for five to ten minutes, or even longer, and is more intense than normal. This is called by Neumann "enduring nystagmus," and is due to loss of the inhibitory control which the cerebellum exercises over the vestibular centers.

Vertigo was present in 56 out of the 86 cases. There is nothing characteristic about cerebellar vertigo. Its intensity is not influenced by looking in different directions, as vestibular vertigo is.

Vomiting occurred in 48 of the cases. It may occur with or without nausea. Vomiting, accompanied by nausea, is due to involvement of the vestibulo-cerebellar tracts.

Of the above-mentioned symptoms, there may be only one or a few present, or there may be none at all.

The cerebellar abscess may be found only at autopsy. Okada found that in ten per cent of cases of cerebellar abscess, death occurred before any symptoms of intracranial involvement appeared.

In fourteen per cent of the cases, the symptoms of cerebellar abscess were obscured by other otitic complications.

In forty-two per cent, the diagnosis was rendered very difficult on account of the presence of other intracranial complications, such as sinus-phlebitis, pachy- or leptomeningitis, or temporo-sphenoidal lobe abscess.

Not all cases of cerebellar abscess which are reported as latent, really are latent, because the cerebellar tests are not always made.

Latent cases are of two kinds:

1. Where there never were symptoms. This might be due to separation of the nervous elements without their destruction. This is more probable in cases of tumor or hemorrhage than with abscess. It may be due to involvement of a silent area of the cerebellum.

2. Where there are symptoms at the beginning, which later disappear. This is due to compensation. Cerebellar symptoms may be made more marked and enduring by the association of lesions in compensatory organs (cerebrum, labyrinth or sensory paths). For instance, in disease of the cerebello-pontine angle, the

eighth nerve is involved along with the cerebellum. This causes very severe symptoms.

The course of cerebellar abscess is usually divided into four stages, the initial stage, the latent stage, the manifest stage and the terminal stage.

The initial stage lasts a day or two and is marked by chills or chilly feelings, and malaise. It is usually not noticed, and is often masked by the symptoms of the ear disease which is the etiological factor.

The latent stage may last anywhere from a few days to many months or even years. It is not really latent, but the symptoms are usually so slight that they are overlooked. There may be occasional headache during this period, with vertigo and loss of appetite.

During the manifest stage, any of the symptoms above described may appear. Sometimes, however, the manifest stage may be entirely wanting, the diagnosis being made at operation or autopsy. The manifest stage may last several weeks.

The terminal stage is the period after the abscess has ruptured either into the fourth ventricle or onto the surface of the cerebellum, and its symptoms are those of the resultant meningitis.

In uncomplicated cases, death may be due to

1. Increasing intracranial pressure with edema of the brain.

2. Encephalitis.

3. Toxic coma.
4. Rupture into the subarachnoid space.
5. Rupture into the fourth ventricle.

Differential Diagnosis

Cerebellar abscess must be differentiated from temporo-sphenoidal lobe abscess, tumor of the cerebellum or of the eighth nerve, labyrinthitis, meningitis, epidural abscess, sinus thrombosis and hysteria.

From temporo-sphenoidal lobe abscess, it is differentiated by the localizing signs. In temporo-sphenoidal lobe abscess, there may be aphasia or hemianopsia. There may be hemianesthesia and hemiparesis of the opposite side. When a hemiparesis is present in cerebellar abscess it is on the side of the lesion.

The disturbances of speech which may be present in cerebellar abscess are disturbances in articulation, in contradistinction to the aphasia which may be present in temporo-sphenoidal lobe abscess.

There may be abnormalities of the ocular muscles in both conditions. Nystagmus is very common in cerebellar abscess, but very rare in temporo-sphenoidal lobe abscess. In cerebellar abscess there may be a spontaneous deviation of one or more extremities, a loss of the reaction movements of the extremities, or a loss of the reaction movements of the trunk.

The location of the headache is not characteristic.

There may be local sensitiveness to percussion over the site of the abscess. Rigidity of the neck is not uncommon with cerebellar abscess.

If the abscess follows labyrinthine disease or sinus thrombosis, it is more apt to be in the cerebellum than in the temporo-sphenoidal lobe.

Cerebellar abscess is differentiated from tumor of the cerebellum by the fact that, in the former condition, there is a middle-ear suppuration. In abscess, there may be some fever, although it is never very high. In tumor, there is a gradual and steady increase in the severity of the symptoms which may extend over a considerable period of time. In abscess, there is usually a period of latency, and then a rapid development of the symptoms, which may remain constant until shortly before death, at which time symptoms of diffuse meningitis supervene. In tumor of the cerebellum the fundus changes are, as a rule, much more marked than in abscess.

In abscess the cerebro-spinal fluid may show an increase in the polynuclear white cells, and bacteria may be present. In tumor, the fluid is more likely to be normal. In the latter the lymphocytes may be increased. With abscess the blood may show increase in the leucocytes.

In tumor of the eighth nerve, the symptoms are similar to those of cerebellar tumor, but deafness is usually one of the first symptoms to appear, and

symptoms of destruction of the eighth and seventh nerves are very prominent.

Cerebellar abscess is differentiated from labyrinthitis by the fact that there is no headache in the latter, but usually a marked headache in the former. The character of the nystagmus is the same in both conditions, but its behavior is different. In cerebellar abscess, the nystagmus may be directed toward the affected side, toward the sound side, or in both directions. It may be rotatory in one direction and horizontal in the opposite direction. In manifest diffuse suppurative labyrinthitis, the nystagmus is always directed away from the diseased ear. In circumscribed labyrinthitis it is usually directed toward the diseased ear.

If the labyrinthine tests show the presence of a diffuse labyrinthitis, i. e., if there is complete deafness and a negative caloric reaction in the ear, and the nystagmus is directed toward the diseased ear, the nystagmus cannot be due to the labyrinthine disease. Provided the opposite ear is normal, it must be retrolabyrinthine in origin, and is probably cerebellar.

In diffuse labyrinthitis, the intensity of the nystagmus steadily diminishes and subsides entirely in two or three weeks. In cerebellar abscess it may remain unchanged for a long time or even become more marked.

Neither the direction of the nystagmus nor its

duration differentiates between cerebellar abscess and circumscribed labyrinthitis. In both conditions, it may be directed toward the diseased side, and may last a long time. Where it is not possible to differentiate between the two conditions, Neumann advises doing a labyrinthectomy. If there was a circumscribed labyrinthitis present, the nystagmus will change to the sound side. If, however, there is a cerebellar abscess present, the nystagmus will remain directed toward the diseased side.

Changes may be present in the cerebro-spinal fluid. The fluid is usually normal in uncomplicated labyrinthine disease.

Spontaneous deviation and loss of reaction movements may be present in cerebellar abscess. In cerebellar abscess, the spontaneous deviation of the extremities and the tendency to fall bear no definite relationship to the spontaneous nystagmus, nor does the direction of the falling change with alterations in the position of the head. With labyrinthine disease, the spontaneous deviations of the extremities and the falling are always in the direction of the slow component of the spontaneous nystagmus, and the spontaneous falling and deviations change with alterations in the position of the head. The reaction movements are present in labyrinthine disease.

Cerebellar abscess is differentiated from meningitis by the following facts: The duration of a cerebellar

abscess is usually weeks or months, that of a meningitis is only days. The temperature in cerebellar abscess is usually low. It may be normal or even subnormal. In meningitis it is usually high. The pulse is usually slow in abscess. In meningitis it is rapid. There is not usually much disturbance of the sensorium in abscess. In meningitis there is marked disturbance of the sensorium, often complete coma. Optic nerve changes are more common in abscess than in meningitis. Focal signs are common in abscess. They are rare in meningitis. Kernig's sign, Babinski's sign, ankle-clonus, increase of the knee-jerks, etc., are not often seen in abscess. The spinal fluid in an abscess may be normal, or may contain an increase in the white cells. In meningitis it contains an increased number of white cells and bacteria. Cerebellar abscess is complicated by meningitis at the end and the symptoms of the latter condition then obscure those of the former.

Tuberculous meningitis resembles abscess more than diffuse purulent meningitis does, because in tuberculous meningitis the temperature is apt to be lower, and there is a tendency to localization. In serous meningitis, the signs of increased intracranial pressure stand in the foreground. There are usually no focal signs. Lumbar puncture discloses a normally constituted fluid under increased pressure.

Epidural abscess is rarely confounded with cere-

bellar abscess. The former is usually found at operation, without being suspected beforehand.

It sometimes causes marked headache, but its symptoms are usually masked by the accompanying mastoid disease. Epidural abscess is often present with cerebellar abscess.

Hysteria may be mistaken for cerebellar abscess. Hysterical patients may have headache and vertigo and even hemiparesis and hemianesthesia; but they have no spontaneous deviations or loss of reaction movements. They have no nystagmus, eye-ground changes, fever, slow pulse or emaciation.

The diagnosis of cerebellar abscess is sometimes made from the findings at the mastoid operation. A fistula may be seen in the dura leading to the cerebellar abscess. There may be discoloration of the dura, or perhaps lack of pulsation of the cerebellum.

When cerebellar abscess is suspected, it is sometimes advisable to do a radical mastoid operation, and expose the dura of the posterior cranial fossa. If there are no local signs in the dura to warrant one in exploring the cerebellum, it is advisable to await developments. If there is no cerebellar abscess present, the symptoms may subside after the operative intervention. If the symptoms persist, the cerebellum can be explored several days later. It is safer to explore the cerebellum at this time, as adhesions

have formed between the dura and pia, and there is less likelihood of infecting the meninges by the exploratory punctures or incisions. However, where there are definite localizing signs of cerebellar abscess, it is not necessary to wait.

CHAPTER V

PROGNOSIS AND TREATMENT OF CEREBELLAR ABSCESS

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CEREBELLAR abscess, if untreated, is almost surely fatal. A few cases of spontaneous cure have been reported in the literature, but such an outcome must be so rare that it should be given no weight whatever in the consideration of the indications for treatment. It is possible that a very small abscess may undergo resorption and thus get well. Those cases of cerebellar abscess which rupture into the mastoid, and from which pus finds exit through a perforation in the drum-membrane or through a fistula in the skin over the outer surface of the mastoid, do not get well. The fistula is too narrow and tortuous to permit adequate drainage of the abscess. From time to time the fistula may become occluded and there is a lighting up of the brain symptoms, and then if there is a re-establishment of drainage, the symptoms may subside.

The only treatment for cerebellar abscess is operation. We can safely say that practically all cases of cerebellar abscess which are not operated upon die.

The percentage of recoveries from cerebellar abscess following operation varies in the hands of different

operators, and after different operative procedures. Macewen, as far back as 1893, reported a series of 19 cases of brain abscess, of which he cured 18 by operation. Of these, 13 were otogenous, 9 being situated in the temporo-sphenoidal lobe and 4 in the cerebellum. No other surgeon has been able to equal this record.

Koerner reported 55 cases of operations for cerebellar abscess in 1901, of which 52.8 per cent were cured.

Heimann collected 519 operations for brain abscess in 1908. Of these 323 were in the cerebrum and 196 in the cerebellum. One hundred and ninety-three (37.08 per cent) got well. Of the healed cases, 144 were in the temporo-sphenoidal lobe, 40 in the cerebellum, and 3 in the temporo-sphenoidal lobe and cerebellum.

Oppenheim and Cassirer collected 76 cases of operations for cerebellar abscess in 1909. Of these 35 (44.9 per cent) were cured.

Dench collected 102 operations for cerebellar abscess, of which 33 recovered. Koch reported 25 cases of operations for cerebellar abscess of which 16 were cured.

Neumann collected 101 cases of cerebellar abscess operated upon, of which 40 recovered.

The results of operation for cerebellar abscess are not as favorable as those for temporo-sphenoidal lobe

abscess. This is due to the fact that it is easier to drain an abscess in the temporo-sphenoidal lobe than in the cerebellum. Furthermore, abscesses in the cerebellum are so close to the respiratory center in the medulla that manipulations in this region can easily cause respiratory paralysis.

In most of the statistics, the percentage of recoveries is probably given too high, since favorable cases are more apt to be reported than unfavorable ones. Furthermore, many of the cases are reported too early. A case should not be reported as cured unless it has remained well for at least six months after the operation; for it is not an uncommon experience for a patient to be apparently well for several months after an operation for cerebellar abscess, and then suddenly develop meningitic symptoms and die.

A patient with cerebellar abscess is never too sick to be operated on. He may be moribund, and yet recover after the operation. Symptoms of meningitis are no contraindication to operation, as sometimes the symptoms of a cerebellar abscess are very similar to those of meningitis.

The causes of death from cerebellar abscess, with or without operation, according to von Bergmann, are as follows:

1. Rupture of the abscess into the fourth ventricle.
2. Rupture onto the surface of the cerebellum with a resultant leptomeningitis.

3. The abscess cannot be found at operation.

4. There is a second abscess.

5. The abscess has more than one chamber.

6. The encephalitis progresses in spite of evacuation of the abscess.

7. Sinus thrombosis with pyemia.

8. Pneumonia.

9. Shock.

10. Respiratory failure.

By far the most common cause of death is purulent leptomeningitis.

The prognosis is most favorable in those cases in which the abscess is near the surface of the cerebellum, especially when there is a fistula through the dura, and when there is a firm capsule about the abscess. The unfavorable cases are those in which the abscess is situated far from the surface and has no capsule.

The prophylactic treatment consists in the treatment of the causes of cerebellar abscess, namely, labyrinthitis, sinus thrombosis and epidural abscess. Suppurative labyrinthitis is treated by means of a radical mastoid operation and labyrinthectomy. Sinus thrombosis is treated by opening the sinus, removing the thrombus, and tying off or resecting the internal jugular vein. Epidural abscess is treated by removing the inner table over the abscess.

Operations on the brain are best done under chloroform anesthesia. Ether causes a congestion of the

brain and increases the tendency to edema of the brain tissue, whereas chloroform diminishes the congestion of the brain. This factor is more important in operations on the cerebellum than in those on the cerebrum, on account of the danger of respiratory paralysis from pressure of the swollen cerebellum upon the medulla. When the operation is done in two stages, the dura being exposed at the first operation, the second stage requires very little anesthesia, as the brain tissue possesses little sensation.

Although the methods of approach may differ in various cases, the treatment of cerebellar abscess must be based upon two principles. The first is the establishment and maintenance of efficient drainage; the second is adequate decompression so that the edema of the brain which threatens the vital centers may be relieved.

In the first place it must be remembered that the vast majority of cerebellar abscesses are situated in the anterior portion of this organ. This is to be expected when one recalls that infection of the cerebellum is usually secondary to a suppurative labyrinthitis, an epidural abscess in the posterior fossa or a thrombo-phlebitis of the sigmoid portion of the lateral sinus.

The operative treatment of cerebellar abscess varies according to the findings. (Figs. 21-23.) The method of approach will materially vary with the

pathway of the brain infection. When a cerebellar abscess is suspected, a simple or a radical mastoid operation is done. In cases of acute middle-ear suppuration, a simple mastoid operation is done, and in chronic middle-ear suppuration, a radical mastoid operation. The inner table is removed, exposing the dura of the posterior fossa.

In all cases, irrespective of the pathway of infection, a wide decompression of the posterior fossa should be done, continuing the removal of bone well down underneath the cerebellum toward the foramen magnum.

If the dura is found to be normal, and the symptoms of cerebellar abscess are only suggested, it is wise to wait, for sometimes the disease in the temporal bone may give symptoms similar to those of intracranial disease, and these symptoms will subside after the mastoid operation.

The exposure of the dura will produce adhesions between the dura and the surface of the cerebellum, so that if the symptoms do not subside after the mastoid operation and it becomes necessary to incise the dura, infection of the meninges is less likely to occur.

Labyrinthine disease is especially liable to simulate cerebellar abscess. If the functional tests show a dead labyrinth, i. e., if there is complete deafness in the affected ear and a negative caloric reaction, and the nystagmus is directed toward the sound side, we may

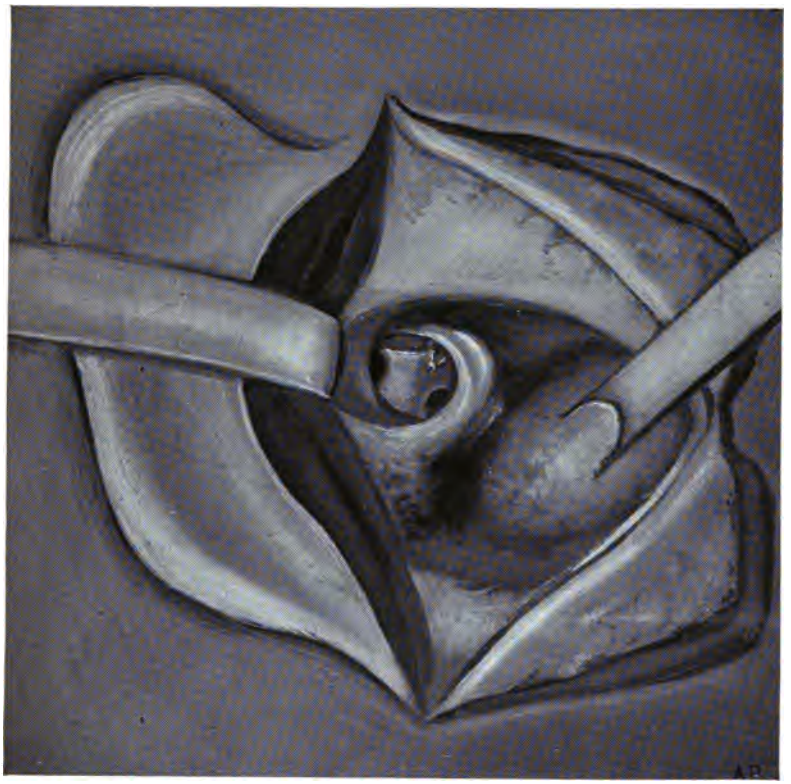

FIG. 21.—OPERATION FOR CEREBELLAR ABSCESS.
A radical mastoid operation has been done. Sinus is being uncovered by chisel.

FIG. 22.—OPERATION FOR CEREBELLAR ABSCESS.
Sinus and cerebellar dura exposed. Incision through dura in front of and behind sinus.

FIG. 23.—OPERATION FOR CEREBELLAR ABSCESS.
Labyrinthectomy. Internal auditory canal exposed. Incision through cerebellar dura in front of sinus.

have either a diffuse suppurative labyrinthitis alone, or a diffuse suppurative labyrinthitis complicated by a cerebellar abscess. A labyrinthectomy is done, and the dura of the posterior fossa exposed. If the dura looks normal, it is not incised unless there be positive signs of cerebellar involvement. The patient is carefully watched, and if the symptoms were due to the labyrinthitis alone, they will improve in a few days. The symptoms of an acute diffuse suppurative labyrinthitis reach their maximum intensity within twenty-four hours, and then show a gradual improvement from day to day. If the symptoms do not clear up in a short while, the cerebellum should be explored for an abscess.

If the functional tests show a dead labyrinth, and the nystagmus is directed toward the diseased side, there is probably a cerebellar abscess complicating a diffuse suppurative labyrinthitis, for a dead labyrinth cannot cause a nystagmus toward its own side. In such a case, the cerebellum is explored immediately.

If the functional tests show a normal labyrinth, a radical mastoid operation is done and the posterior fossa exposed. If an epidural abscess is found and a fistula leading into a cerebellar abscess, the dura is first cleaned thoroughly with saline followed by alcohol. Michaelsen advises mopping the dura with tincture of iodin. After the dura is thoroughly cleaned,

a knife is introduced into the fistula and the abscess slit open for three or four centimeters.

If the dura of the posterior fossa is normal, it is sometimes advisable to expose the dura of the middle fossa, for temporo-sphenoidal lobe abscess occasionally gives rise to symptoms very similar to those of cerebellar abscess. If nothing abnormal is found here, the cerebellum is explored. The cerebellum should never be explored through an intact dura, for the following reasons:

1. The exploring needle or knife may wound a meningeal vessel of some size and cause a subdural hemorrhage, which, finding no escape, may cause dangerous pressure upon the cerebellum.

2. If the abscess is found, some pus may follow the needle, as it is withdrawn, and, flowing into the subdural space, give rise to a diffuse meningitis.

3. If the needle is plunged through a pathologically thickened and inflamed dura, it may carry some of the bacteria which are lodged in the deeper layers of the dura into the brain tissue.

It is best to incise the dura before exploring the cerebellum. The point at which the cerebellum is first explored is Trautmann's triangle. This is a triangle whose upper margin is formed by the superior petrosal sinus, its posterior margin by the anterior margin of the lateral sinus, and its anterior margin by the posterior semicircular canal. The apex of this

triangle is directed downward and is in the region of the saccus endolymphaticus, and the base of the triangle is directed upward. A hollow needle or a knife or a pair of forceps may be used to explore the cerebellum. The disadvantages of a hollow needle are that the contents of the abscess may be so thick that they cannot be drawn through the canula, or the lumen may become obstructed by a piece of brain tissue before it reaches the abscess. The disadvantages of a knife are that a greater extent of brain tissue is injured than with a needle, and there is greater likelihood of injuring a large vessel. With blunt forceps, there is less danger of bleeding. On the whole, the knife has seemed the most reliable instrument to us. After the abscess is found, the incision may be dilated by means of forceps. If a needle is used, leave the needle in situ when the abscess is found, and run the knife along this as a guide.

Before the drainage tube is introduced into the abscess a free dural incision is made in order to attain sufficient decompression. A linear or semicircular incision is preferable to a cross incision, as the latter is conducive to brain hernia.

The needle or knife is passed through Trautmann's triangle into the cerebellum in various directions, until the abscess is discovered. It should not be allowed to pass in further than two and one-half to three cm., in order to avoid entering the fourth ventricle. The

blade of the knife or the needle may be marked off in centimeters. If the abscess is not found in Trautmann's triangle, the cerebellum is explored behind the lateral sinus. The cerebellar hemisphere is widely exposed here by the removal of a portion of the occipital bone, and the dura is incised. Krause advises making a U-shaped flap in the dura, and packing off the subdural space by means of a strip of gauze at the edges of the dural incision. In this way every drop of pus which passes out of the cerebellar incision can be mopped up, and there is very little danger of causing a diffuse purulent meningitis.

The area through which the searcher is being passed should be protected by pledgets of cotton wet with warm saline. If a canula be used, when the abscess is found and pus makes its appearance at the proximal end of the canula, it may be sucked away by means of a suction apparatus. It must be understood that suction should not be applied through the canula. Merely the drops of pus as they flow out of the mouth of the canula should be sucked away. In this way the field does not become soiled. Forcible aspiration of the contents of the abscess is not devoid of danger.

When the abscess is found and the pus evacuated, a drainage tube is inserted into the cavity. It is not advisable to wash out the abscess cavity nor to introduce the finger, or encephaloscope, as such interventions increase the encephalitis. It seems that the cere-

bellum will bear less handling than the cerebrum. For drainage material, rubber tubing or a decalcified chicken-bone tube seems to be the best. Uffenorde and Michaelsen use a glass tube, with a gauze wick inside. Cigarette drains or gauze packing do not seem to be very effective, as they cause a retention of secretions. It is advisable to have several perforations at the sides of the tube in case the end of the tube becomes blocked by pieces of necrotic tissue. The outer end of the tube is surrounded by iodoform gauze, and a dressing applied. It may be necessary to hold the tube in place by means of a safety-pin passed through it. The dressings are changed every day or even oftener, depending upon the amount of secretion. The tube is gradually shortened as the abscess diminishes in size, and is finally left out altogether.

Some otologists prefer to explore the cerebellum through the occipital bone instead of through the mastoid. In this way they avoid carrying infection into the cerebellum from the infected mastoid cavity. However, this advantage is overweighed by several disadvantages, viz.:

1. Cerebellar abscesses are usually located near the median portion of the ventral surface of the cerebellum, and are reached most easily through the mastoid.

2. Exposure of the cerebellar dura through the

mastoid often gives valuable information as to the presence of an abscess.

3. The mastoid operation removes the primary cause of the cerebellar infection.

The operation through the occiput is done as follows (Fig. 24): An inverted U-shaped incision is made through the skin and the periosteum over the occiput. The anterior limb of this incision extends along the posterior margin of the mastoid, the upper limb along the external occipital crest and the posterior limb along the middle line of the skull down to the root of the neck. The periosteum is reflected from the bone. A trephine opening is made through the middle of the exposed bone, and the opening enlarged by means of a rongeur forceps until the lateral sinus is seen above, the sigmoid sinus laterally, and the occipital sinus mesially. A U-shaped dural flap is now made parallel to the edges of the bone wound, and the subdural space packed off by means of a strip of iodoform gauze which is inserted between the dura and the surface of the cerebellum. The cerebellum is then explored for the abscess with a knife, grooved director, or needle.

In some cases, when the abscess is opened through an incision in Trautmann's triangle, a counter opening may be made behind the lateral sinus, and through and through drainage instituted. When the lateral sinus is thrombosed, it is opened, and an incision made

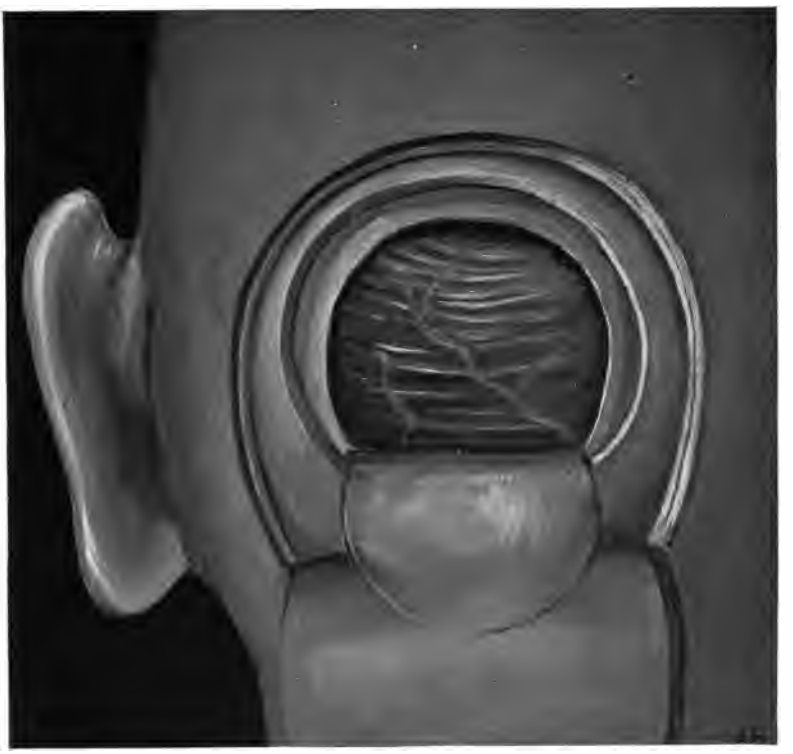

FIG. 24.—OPERATION FOR CEREBELLAR ABSCESS.
Cerebellum uncovered from behind.

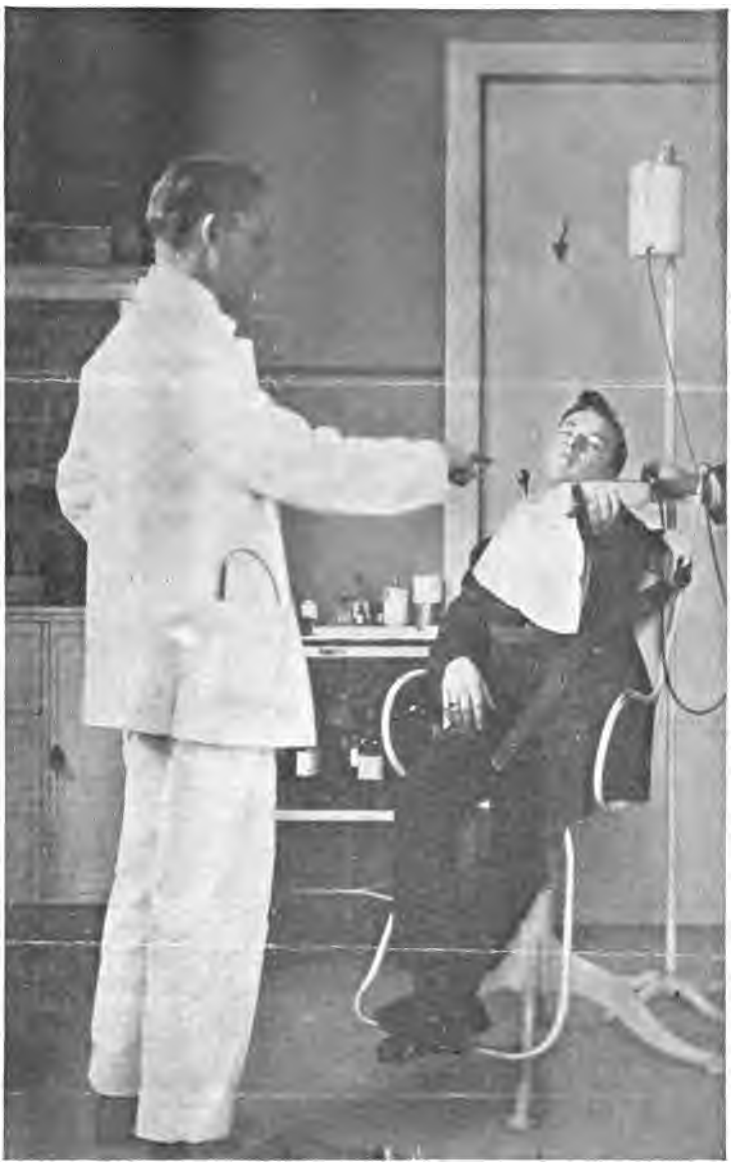

FIGURE 18.–Pointing Reaction with Caloric Vestibular Stimulation.

CEREBELLAR ABSCESS

ITS ETIOLOGY, PATHOLOGY, DIAGNOSIS AND TREATMENT

INCLUDING ANATOMY AND PHYSIOLOGY OF THE CEREBELLUM

BY

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through the inner wall of the sinus. In this way very free access can be gotten to the cerebellum.

The following procedure has been suggested by us,[1] and while we have had no personal experience with it, Bourguet, in a single case of cerebellar abscess, has carried it to a successful issue. The procedure entails the sacrifice of the lateral sinus and while this must be carefully considered, it seems to us that in a complication as formidable as cerebellar abscess, the advantages gained by such a sacrifice make it well worth while.

The cerebellar dura is exposed in front of and behind the sinus. A small incision is made in the dura in front of the sigmoid sinus just below the knee. Through this incision an aneurysm needle threaded with chromic gut is passed, from before backward, around the sinus, until the blunt threaded point presents underneath the dura behind the sinus. The dura on the point of the aneurysm needle is then incised and the needle pushed through.

The chromic gut is then tied. A similar ligature is placed around the sinus as close as possible to its horizontal portion. The outer wall of the sinus between the ligatures is opened and cut away. Such an exposure affords an ideal approach to collections of pus within the cerebellum.

[1] Since the above was written we have learned, through a private communication from Dr. Edward B. Dench, that Mr. Ballance, of London, has also successfully used this procedure.

Koerner gives the following statistics of 139 cases of brain abscess operated upon by various methods:

	Cure.	Death.
1. Abscess opened through diseased temporal bone	22	14
2. Abscess opened through diseased temporal bone with counter opening through outside	4	0
3. Abscess opened through fistula on outer surface of skull	4	0
4. Abscess opened through outer wall of skull	42	52
5. Method not given	0	1

The after treatment of cerebellar abscess consists of absolute rest in bed. Everything should be avoided which causes hyperemia of the brain. The amount of secretion, the temperature and the pulse are the guides for the frequency of the dressings. If symptoms indicate retention of secretions, the dressings must be changed and the drainage tube inspected and cleaned out. It may be necessary to search for a second abscess or for a pocket in the first abscess.

If a radical mastoid operation was done, a secondary plastic should be made only after the abscess is entirely healed.

The commonest complication following operation for cerebellar abscess is prolapse of the brain. This is due to encephalitis, to the fact that the abscess was

not found, or that a pocket remains which was not opened, or that there is a second abscess.

When it is due to an unopened pocket or a second abscess, it recedes after evacuation of the pus. If no pus is found, an attempt is made to cause it to recede by means of a firm pressure bandage. This is successful in most cases, although in extreme cases it may take several weeks. If this is not successful it may be necessary to excise the prolapse. As the prolapse consists largely of edematous tissue, and contains very little normal brain tissue, this intervention is not as serious a one as may appear. The prolapse may be made to recede by doing a lumbar puncture. After a successful operation for cerebellar abscess, the improvement is immediate. The sensorium improves. The pulse increases rapidly. The paralytic signs usually clear up. Some of the symptoms may remain permanently. These are the symptoms which are due to actual destruction of tissue. Cysts and cicatrices may remain after operation and give rise to attacks of epilepsy.

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